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ORIGINAL ARTICLE

Early predictors of dysphagia in ischaemic stroke patients

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Abstract

Background and purpose: Post-stroke dysphagia affects outcome. In acute stroke patients, the aim was to evaluate clinical, cognitive and neuroimaging features associated with dysphagia and develop a predictive score for dysphagia.

Methods: Ischaemic stroke patients underwent clinical, cognitive and pre-morbid function evaluations. Dysphagia was retrospectively scored on admission and discharge with the Functional Oral Intake Scale.

Results: In all, 228 patients (mean age 75.8 years; 52% males) were included. On admission, 126 (55%) were dysphagic (Functional Oral Intake Scale ≤6). Age (odds ratio [OR] 1.03, 95% confidence interval [CI] 1.00–1.05), pre-event modified Rankin scale (mRS) score (OR 1.41, 95% CI 1.09–1.84), National Institutes of Health Stroke Scale (NIHSS) score (OR 1.79, 95% CI 1.49–2.14), frontal operculum lesion (OR 8.53, 95% CI 3.82–19.06) and Oxfordshire total anterior circulation infarct (TACI) (OR 1.47, 95% CI 1.05–2.04) were independently associated with dysphagia at admission. Education (OR 0.91, 95% CI 0.85–0.98) had a protective role. At discharge, 82 patients (36%) were dysphagic. Pre-event mRS (OR 1.28, 95% CI 1.04–1.56), admission NIHSS (OR 1.88, 95% CI 1.56–2.26), frontal operculum involvement (OR 15.53, 95% CI 7.44–32.43) and Oxfordshire classification TACI (OR 3.82, 95% CI 1.95–7.50) were independently associated with dysphagia at discharge. Education (OR 0.89, 95% CI 0.83–0.96) and thrombolysis (OR 0.77, 95% CI 0.23–0.95) had a protective role. The 6-point "NOTTEM" (NIHSS, opercular lesion, TACI, thrombolysis, education, mRS) score predicted dysphagia at discharge with good accuracy. Cognitive scores had no role in dysphagia risk.

Conclusions: Dysphagia predictors were defined and a score was developed to evaluate dysphagia risk during stroke unit stay. In this setting, cognitive impairment is not a predictor of dysphagia. Early dysphagia assessment may help in planning future rehabilitative and nutrition strategies.

KEYWORDS
dysphagia, evaluation, prognosis, stroke

INTRODUCTION

The incidence of dysphagia in patients with ischaemic stroke is highly variable, depending on assessment methods and ranging from 8% to 80% [1–3]. The frequency is higher (between 64% and 80%) when instrumental tools (videofluoroscopy or fiberoptic endoscopic evaluation of swallowing [FEES]) are used [1, 2]. Dysphagia has a negative impact on prognosis, length of hospital stay, aspiration pneumonia incidence and mortality [4–6]. Reported pneumonia incidence amongst dysphagic patients ranges between 16% and 33% [1], and 14.5% of ischaemic stroke patients show signs of lower airway infection [7, 8]. Pneumonia incidence is sevenfold higher in ischaemic stroke patients with aspiration compared with ischaemic stroke patients without aspiration [9]. A complete or partial impairment in swallowing causes also a reduced intake of food, a progressive weight loss and a nutrient deficiency, leading to protidic-energetic malnutrition, a factor independently associated with long-term worse prognosis in stroke patients [10]. Therefore, rapid and reliable dysphagia risk stratification tools could be important to identify acute stroke patients to be referred to speech therapist and phoniatric evaluation, prevent infective complications, reduce malnutrition risk and better plan naso-gastric tube (NGT) management and percutaneous endoscopic gastrostomy (PEG) placement [4].

So far, little attention has been paid to the possible impact that cognitive impairment may have on dysphagia recovery in the acute phase of stroke. Because cognitive impairment is strongly associated with stroke and represents one of its main complications [11], it seems potentially useful to explore also its association with dysphagia persistence early after stroke.

The aims of this study were (i) to evaluate clinical, including measures of cognitive impairment, and neuroimaging characteristics associated with dysphagia (defined as a Functional Oral Intake Scale [FOIS] ≤6) and their recovery in the acute phase of stroke (i.e., during the stay in a stroke unit); (ii) to develop an easy-to-use score, applicable at the bedside, for predictive and prognostic purposes concerning dysphagia in acute stroke patients.

METHODS

Study population

Data were retrospectively analyzed from all patients suffering from ischaemic stroke and consecutively admitted to the Stroke Unit of the Luigi Sacco Hospital, Milan, between 1 January 2018 and 31 May 2019, whose diagnosis was formulated by a stroke neurologist based on clinical and neuroimaging data. Patients with a final diagnosis of transient ischaemic attacks, hemorrhagic stroke, cerebral venous thrombosis or stroke mimics were excluded from the analysis to increase homogeneity between patients regarding admission diagnosis.

Information about the following demographic and clinical variables were collected: sex, age, education (expressed in years of schooling) and cardiovascular risk factors (hypertension by American College of Cardiology 2017 criteria, atrial fibrillation by European Society of Cardiology 2016 criteria, diabetes mellitus by World Health Organization criteria, past or current smoker status, weight and body mass index, hypercholesterolemia and hypertriglyceridemia by European Society of Cardiology 2019 criteria) [12–15]. The clinical characteristics of ischaemic stroke were assessed in terms of severity (National Institutes of Health Stroke Scale, NIHSS) [16], site of infarct (side and location, i.e., cortical, subcortical, cerebellar or brainstem), affected vascular territories according to the Oxfordshire classification of stroke (partial anterior circulation infarct, PACI; total anterior circulation infarct, TACI; posterior circulation infarct; lacunar infarct) [17]. The administration of acute phase treatments (intravenous thrombolysis and/or endovascular thrombectomy) was also recorded.

Cognitive measures

A simple cognitive evaluation was performed in the stroke unit with the Montreal Cognitive Assessment—Basic version (MoCA-B) test [18] and the Clock Drawing Test (CDT) [19].

The presence of pre-stroke cognitive impairment and/or behavioral disorders were assessed with the Clinical Dementia Rating (CDR) global score and the Neuropsychiatric Inventory Questionnaire (NPI-Q), administered to the patient's caregiver during the stroke unit stay. CDR is a widely used scale in the evaluation of mild cognitive impairment and dementia and includes six domains (memory, orientation, judgment and problem solving, community affairs, home and hobbies, personal care) that are evaluated in a semi-quantitative way by an informant. The level of impairment for each domain ranges from 0 (no impairment) to 3 (severe impairment). Final computation of the CDR global score was made as suggested by Hughes et al. [20]. A global score of 0 was considered suggestive of the absence of a previous cognitive impairment; a global score of 0.5 suggestive of a previous mild cognitive impairment; a global score of ≥1 suggestive of a previous major cognitive impairment (dementia). NPI is a scale developed to assess psychopathological features in dementia patients; it evaluates 12 common neuropsychiatric disturbances in dementia: delusions, hallucinations, agitation, depression, anxiety, apathy, irritability, euphoria, disinhibition, aberrant motor behavior, night-time behavior disturbances, and appetite and eating abnormalities. The severity of each neuropsychiatric symptom is rated during an interview with the patient's caregiver. NPI also assesses the amount of caregiver distress engendered by each neuropsychiatric symptom [21]. To quantify the impact of cognitive impairment on daily life activities, the activities of daily living (ADL) [22] and instrumental activities of daily living (IADL) scales [23] were used.

Outcome measures

Length of stay, etiological classification of stroke (according to the "Trial of ORG 10172 in Acute Stroke Treatment"—TOAST

classification), modified Rankin scale (mRS), NIHSS and Barthel index score at discharge were registered [16, 24–26]. Destination of discharge (home, rehabilitation or nursing home) were also noted. Any death during hospitalization was registered.

Dysphagia assessment

At stroke unit entry, all patients underwent dysphagia screening [27]. For the purpose of this study, data from all patients were reviewed by one author (DM) who retrospectively applied the FOIS [28–30], a validated scale based on the type of oral intake. The scale ranges from 1 (no oral intake) to 7 (total oral intake with no restrictions) (Table S1). For the present study, the FOIS score was retrospectively assigned based on the results of the speech therapist's and neurologist's evaluations carried out during the hospital stay and on the food characteristics prescription. In this study, the presence of dysphagia was defined as an FOIS score ≤6.

Neuroimaging evaluation

For the evaluation of neuroimaging, routine examinations performed during the stroke unit stay for clinical purposes were used. When available, 1.5 T magnetic resonance imaging (MRI) was preferred to computed tomography (CT) for this evaluation. Affected cerebral vascular territory (anterior, middle, posterior or vertebro-basilar artery territory) and the distribution of ischaemic lesions (cortical, subcortical, cerebellar or brainstem) were assessed. Considering the location of the acute lesion, in this study the focus was on the presence of frontal operculum involvement, assessed by visual assessment of CT or MRI scans by one author (DM), because this location has recently been found to be of particular relevance for dysphagia [31]. The neuroimaging assessment was done in a blind manner to the clinical information. The following visual scales were used: two scales for leukoaraiosis (van Swieten et al. [32] and Fazekas et al. [33] scales); the Global Cortical Atrophy Scale, a qualitative visual scale for global atrophy evaluation in 13 brain regions, both deep and superficial [34]; the Scheltens et al. visual scale for medial temporal atrophy evaluation [35].

Statistical analysis

Demographic, clinical, neuroimaging and cognitive variables were compared between dysphagic and non-dysphagic patients using the Fisher exact test and the chi-squared test for categorical variables and the Mann–Whitney test for continuous variables. The same tests were used to compare the groups of patients who recovered or not from dysphagia at discharge. All analyses were conducted using the MedCalc statistical analysis software, version 19.1, choosing a p value of 0.05 as a threshold for significance. In addition, for variables with a significant p value, odds ratios (ORs) were calculated with the respective 95% confidence intervals (CIs), both upon entering the stroke unit and at discharge.

A logistic regression based multivariate analysis was performed to explore differences between dysphagic and non-dysphagic patients, including data emerging from the univariate analysis. Variables associated with dysphagia at discharge in the multivariate analysis were included in a point-based score, suitable for use in the individual patient on stroke unit admission. Cut-offs for relevant continuous variables were determined examining different variable distributions between dysphagic and non-dysphagic patients using box and whisker plots. The attribution of item points for the score was assigned considering the strength of the association underlined by the multivariate analysis (no points for OR ≤1, 1 point for OR between 1 and 2, 2 points for OR ≥2). The score was assessed in terms of sensitivity, specificity, and positive and negative predictive value, through receiver operating characteristic (ROC) curve evaluation.

RESULTS

In all, 228 patients (mean age 75.8 ± 12.4 years; 52% males) were included. The median length of hospital stay was 9 days (interquartile range 6–13).

Dysphagia on stroke unit admission

The evaluation of the data of the 228 patients admitted to the stroke unit showed that 126 (55%) had some degree of dysphagia (FOIS ≤6). Of these patients, 34 (27% of dysphagic patients and 15% of the patients admitted to the stroke unit) had severe dysphagia (FOIS ≤3), with need for NGT placement.

Demographic, clinical (including cognitive) and neuroimaging variables of dysphagic and non-dysphagic patients are summarized in Table 1.

On stroke unit admission, compared to non-dysphagic patients, dysphagic patients were older, had a lower education level, higher pre-event mRS, more severe stroke and more frequently a TACI stroke. The involvement of the frontal operculum (regardless of the side) was significantly more frequent in dysphagic than in non-dysphagic patients. The other neuroimaging variables were not significantly associated with dysphagia.

Gender, pre-event cognitive and functional status (CDR, NPI, ADL, IADL), coexistence of vascular risk factors, nutritional variables, MoCA-B and CDT scores, acute phase therapies, cortical or subcortical localization, vascular territory or affected side were not significantly different between the two groups of patients.

The presence of dysphagia was associated with a longer hospitalization, higher mRS and NIHSS scores at discharge, a lower Barthel index score, and an increased risk of discharge towards rehabilitation or nursing homes. The two intra-hospital deaths occurred within the group of dysphagic patients and were due to aspiration pneumonia.

TABLE 1 Demographic, clinical and neuroimaging characteristics of patients according to the presence/absence of dysphagia on stroke unit admission.

	All, $N = 228$	Non-dysphagic, $N = 102$ (45%)	Dysphagic, $N = 126$ (55%)	Univariate analysis
Sex, *n* (%)				
Female	109 (48%)	42 (41%)	67 (53%)	ns
Male	119 (52%)	60 (59%)	59 (47%)	
Age, years, mean (SD)	75.8 (12.4)	72.1 (13.5)	78.9 (10.4)	$p < 0.001$
Education, years, median (IQR)	8 (5–11)	8 (6–13)	7.5 (5–8)	$p < 0.001$
mRS pre-event, median (IQR)	0 (0–1)	0 (0–0)	0 (0–2)	$p < 0.001$
Lost ADL, median (IQR)	0 (0–1)	0 (0–0)	0 (0–1)	ns
Lost IADL, median (IQR)	0 (0–1)	0 (0–1)	0 (0–2)	ns
CDR, median (IQR)	0 (0–0.5)	0 (0–0.5)	0 (0–0.5)	ns
NPI, median (IQR)	1 (0–3)	1 (0–3)	1 (0–3)	ns
Vascular risk factors				
Hypertension, *n* (%)	183 (80%)	85 (83%)	98 (78%)	ns
Atrial fibrillation, *n* (%)	66 (29%)	30 (29%)	36 (29%)	ns
Diabetes, *n* (%)	54 (24%)	25 (25%)	29 (23%)	ns
Smoking, *n* (%)	107 (47%)	53 (52%)	54 (43%)	ns
BMI class, *n* (%)				
Underweight	4 (2%)	2 (2%)	2 (2%)	ns
Normal	81 (36%)	38 (37%)	43 (34%)	
Overweight	87 (38%)	40 (39%)	47 (37%)	
Obese	35 (15%)	15 (15%)	20 (16%)	
Hypercholesterolemia, *n* (%)	148 (64%)	65 (64%)	83 (66%)	ns
Hypertriglyceridemia, *n* (%)	11 (5%)	4 (4%)	7 (6%)	ns
NIHSS, median (IQR)	4 (2–8)	2 (1–4)	7 (4–14)	$p < 0.001$
MoCA-B, median (IQR)	23 (18–26)	23 (18–25)	23 (18–26)	ns
CDT, median (IQR)	9 (6–12)	9 (6–12)	9 (7–11)	ns
Thrombolysis, *n* (%)	44 (19%)	16 (20%)	28 (22%)	ns
Thrombectomy, *n* (%)	23 (10%)	7 (10%)	16 (13%)	ns
Imaging modality, *n* (%)				
CT	91 (39%)	35 (34%)	56 (44%)	ns
MRI	4 (2%)	2 (2%)	2 (2%)	
Both	133 (58%)	65 (63%)	68 (54%)	
Infarct laterality, *n* (%)				
Right	83 (36%)	41 (40%)	42 (33%)	ns
Left	129 (57%)	57 (56%)	72 (57%)	
Bilateral	16 (0.1%)	4 (4%)	12 (10%)	
Infarct location, *n* (%)				
Cortical	121 (53%)	52 (51%)	74 (59%)	ns
Subcortical	89 (39%)	40 (39%)	46 (36%)	
Cerebellar	10 (4%)	6 (6%)	4 (3%)	
Brainstem	6 (3%)	4 (4%)	2 (2%)	
Arterial territory, *n* (%)				
ACA	2 (1%)	1 (1%)	1 (1%)	ns
MCA	196 (86%)	85 (83%)	111 (88%)	
PCA	14 (6%)	6 (6%)	8 (6%)	
VB	16 (7%)	10 (10%)	6 (5%)	

(Continues)

TABLE 1 (Continued)

	All, $N = 228$	Non-dysphagic, $N = 102$ (45%)	Dysphagic, $N = 126$ (55%)	Univariate analysis
Oxfordshire class, *n* (%)				
LACI	41 (18%)	18 (18%)	23 (18%)	TACI vs. all: $p < 0.01$
PACI	138 (61%)	67 (66%)	71 (56%)	
POCI	25 (10%)	16 (16%)	9 (7%)	
TACI	24 (11%)	1 (1%)	23 (18%)	
Frontal operculum involvement, *n* (%)				
Opercular lesion	61 (27%)	8 (10%)	53 (42%)	$p < 0.001$
Right opercular	20 (9%)	1	19	
Left opercular	41 (18%)	7	34	
White matter lesions				
van Swieten scale, median (IQR)	2 (1–4)	2 (1–4)	2 (1–4)	ns
Fazekas scale, median (IQR)	2 (1–4.5)	3 (1–5)	2 (1–4)	ns
Cortical atrophy				
Global Cortical Atrophy Scale, median (IQR)	15 (8–20)	15 (9–20)	14 (8–21)	ns
Scheltens scale, median (IQR)	2 (0–2)	2 (0–2,5)	2 (0–2)	ns
Stroke etiology, *n* (%)				
LAA	60 (24%)	26 (25%)	34 (27%)	ns
SVO	55 (24%)	25 (25%)	30 (24%)	
CE	62 (27%)	27 (26%)	35 (28%)	
Other	4 (2%)	3 (3%)	1 (1%)	
Undetermined	40 (17%)	19 (19%)	21 (17%)	
Multifactorial	7 (3%)	2 (2%)	5 (4%)	
Swallowing tests				
FOIS, median (IQR)	6 (4–7)	7 (7–7)	4 (4–6)	$p < 0.001$
Stroke outcome at discharge				
mRS, median (IQR)	1 (0–4)	0 (0–1)	3 (1–4)	$p < 0.001$
NIHSS, median (IQR)	2 (0–4)	1 (0–1)	3 (1–7)	$p < 0.001$
Barthel, median (IQR)	90 (49–100)	100 (92–100)	60 (25–100)	$p < 0.001$
Length of hospital stay, days, median (IQR)	9 (6–13)	7 (5–10)	10 (8–15)	$p < 0.001$
Discharge (destination)				
Home	130 (57%)	87 (85%)	43 (34%)	$p < 0.001$
Rehabilitation	68 (30%)	12 (12%)	56 (44%)	
Nursing home	28 (12%)	3 (3%)	27 (21%)	
Death	2 (0.1%)	0	2 (0.1%)	

Abbreviations: ACA, anterior cerebral artery; ADL, activities of daily living; BMI, body mass index; CDT, Clock Drawing Test; CE, cardioembolic; CDR, Clinical Dementia Rating scale; CT, computed tomography; FOIS, Functional Oral Intake Scale; IADL, instrumental activities of daily living; IQR, interquartile range; LAA, large artery atherosclerosis; LACI, lacunar cerebral infarct; MCA, middle cerebral artery; MoCA-B, Montreal Cognitive Assessment—Basic; MRI, magnetic resonance imaging; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; NPI, Neuropsychiatric Inventory; PACI, partial anterior cerebral infarct; PCA, posterior cerebral artery; POCI, posterior cerebral infarct; SVO, small vessel occlusion; TACI, total anterior cerebral infarct; VB, vertebrobasilar.

In the multivariate analysis model, age (OR 1.03, 95% CI 1.00–1.05; $p < 0.05$), pre-event mRS (OR 1.41, 95% CI 1.09–1.84; $p < 0.001$), NIHSS at onset (OR 1.79, 95% CI 1.49–2.14; $p < 0.0001$), frontal operculum involvement (OR 8.53, 95% CI 3.82–19.06; $p < 0.0001$) and Oxfordshire classification of stroke (TACI vs. other types, OR 1.47, 95% CI 1.05–2.04; $p < 0.05$) were independently associated with an increased risk of acute phase dysphagia. By contrast, education was found to have a slight independent protective effect on dysphagia development in the acute stroke phase (OR 0.91, 95% CI 0.85–0.98; $p < 0.001$).

Dysphagia at discharge

Out of the 128 dysphagic patients at admission, 43 (34%) recovered from dysphagia, 83 (66%) did not recover, and two dysphagic patients died from pneumonia during hospitalization.

Variables associated with dysphagia recovery during stroke unit stay in the univariate analysis were baseline NIHSS, Oxfordshire non-TACI type of stroke, frontal operculum sparing. Thrombolysis and thrombectomy treatments were more common in the group of patients with persistent dysphagia at stroke unit discharge (Table 2).

In the multivariate analysis, variables associated with dysphagia at discharge in dysphagic patients at admission were baseline NIHSS (OR 1.59, 95% CI 1.26–1.99) and frontal operculum involvement (OR 4.52, 95% CI 1.24–16.54).

After a median length of stay of 9 days (interquartile range 6–13), dysphagia was observed in 82 patients of 226 (36% of the total). Of these patients, 24 (24% of dysphagic patients and 11% of the total) had severe dysphagia, implying an NGT positioning in 20 and PEG positioning in four patients with a severe swallowing impairment.

Demographic, clinical, cognitive and neuroimaging variables of patients with and without dysphagia at discharge are summarized in Table 3.

At discharge, compared to non-dysphagic patients, dysphagic patients had a significantly higher age, lower education and higher mRS. Moreover, patients with dysphagia at discharge had more severe strokes than non-dysphagic patients, and more frequently TACI strokes. On the other hand, patients without dysphagia at discharge more frequently suffered a stroke classified as PACI.

In the univariate model, patients with TACI had an increased risk of dysphagia compared to those with other stroke distributions (OR 55.75, 95% CI 7.36–422.33). Patients with PACI had a reduced risk of dysphagia compared to patients with other stroke distributions (OR 0.44, 95% CI 0.25–0.77). The involvement of the frontal operculum (regardless of the side) was more frequent in patients with dysphagia at discharge, conferring an increase in the risk of dysphagia at discharge up to 15 times greater than that of patients without frontal operculum involvement. Other neuroimaging variables were not significantly associated with dysphagia at discharge.

A protective role of thrombolytic treatment against dysphagia at discharge from the stroke unit was found (OR 0.39, 95% CI 0.20–0.76).

A statistically significant association between obesity and failure to recover a normal swallowing function was found (OR 2.41, 95% CI 1.2–4.8). In contrast, a protective role of normal weight was detected (OR 0.50, 95% CI 0.28–0.89). Other factors analyzed were not significantly differently distributed between dysphagic and non-dysphagic patients.

Multivariate analysis results partially confirmed those of the univariate analysis, showing that pre-event mRS (OR 1.28, 95% CI 1.04–1.56; $p < 0.001$), NIHSS at stroke onset (OR 1.88, 95% CI 1.56–2.26; $p < 0.0001$), frontal operculum involvement (OR 15.53, 95% CI 7.44–32.43; $p < 0.0001$) and Oxfordshire classification (TACI vs. other types, OR 3.82, 95% CI 1.95–7.50; $p < 0.0001$) were independently associated with an increased risk of dysphagia at discharge from the stroke unit. In contrast, education (OR 0.892, 95% CI 0.83–0.96; $p < 0.001$) and intravenous thrombolysis (OR 0.77, 95% CI 0.23–0.95; $p < 0.05$) were protective factors against dysphagia at discharge.

Based on the results of the multivariate analysis, a score was developed called NOTTEM (acronym for NIHSS, operculum, TACI, thrombolysis, education, mRS), calculated as shown in Table 4. The score has values ranging from a minimum of 0 (absence of all risk factors and occurrence of all protective factors against dysphagia at discharge) to a maximum of 9 (absence of all protective factors and presence of all risk factors for dysphagia at discharge).

A NOTTEM score >3 has good sensitivity and specificity in predicting dysphagia at discharge (after a median time of 9 days), as shown by the ROC curve (area under the curve 0.88, 95% CI 0.83–0.92; sensitivity 73.2%, 95% CI 62.2–82.4; specificity 87.5%, 95% CI 81.0–92.4; positive predictive value 76.9, 95% CI 68.0–84.0; negative predictive value 85.1, 95% CI 79.9–89.2) (Table 5, Figure 1).

DISCUSSION

In this study, it was possible to find variables associated with dysphagia at stroke unit discharge and to develop a score predictive of dysphagia based on clinical and neuroimaging variables assessed at stroke unit admission.

Dysphagia is a stroke complication with heavy prognostic implications [1–3]. Its presence at admission and persistence are independent predictors of poor outcome, favoring the onset of dysphagia-related complications, like aspiration pneumonia and malnutrition [1–3]. Hence, early detection and management of dysphagia leads to fewer complications, a shorter hospitalization and ultimately a better prognosis [1–3].

The management of dysphagia since the very early hospitalization days is of utmost importance to assure a better outcome. In this regard, the establishment of changes in the rheological characteristics of foods or, in the cases of more severe dysphagia, the early start of enteral nutrition with NGT or PEG are strategic in the management of a stroke patient [4, 5]. On the other hand, NGT or PEG positioning are interventions with potential short- and long-term complications [4, 6]. It is therefore important to develop clinical indicators capable of predicting, with a good degree of accuracy, the exact timing for positioning a device for nutrition, also trying to predict in which type of patient this intervention is not indicated, given the associated risks.

Other studies with a perspective design, different screening modalities and a slightly different population (younger patients with lower NIHSS at admission) evaluated prevalence, risk factors and complications of post-stroke dysphagia [36].

Other studies identified factors influencing the severity of dysphagia: age of patient, extent of ischaemic lesion, male sex, frontal opercular involvement and nutritional status [36–49]. Some of these studies also identified prognostic scores, like the "predictive

TABLE 2 Demographic, clinical and neuroimaging characteristics of dysphagic patients at stroke unit admission according to the presence/absence of dysphagia at discharge.

	Dysphagic at admission, *N* = 126 (2 deaths)	Non-dysphagic at discharge, *N* = 43 (34%)	Dysphagic at discharge, *N* = 83 (66%)	Univariate analysis
Sex, *n* (%)				
Female	67 (53%)	20 (46%)	39 (47%)	ns
Male	59 (47%)	23 (54%)	44 (53%)	
Age, years, mean (SD)	78.9 (10.4)	78.6 (10.7)	79.1 (10.3)	ns
Education, years, median (IQR)	7.5 (5–8)	8 (5–8)	6 (5–8)	ns
mRS pre-event, median (IQR)	0 (0–2)	0 (0–3)	0 (0–2)	ns
Lost ADL, median (IQR)	0 (0–1)	0 (0–0)	0 (0–1)	ns
Lost IADL, median (IQR)	0 (0–2)	0 (0–1.5)	0 (0–2)	ns
CDR, median (IQR)	0 (0–0.5)	0 (0–0)	0 (0–0.5)	ns
NPI, median (IQR)	1 (0–3)	1 (0–4)	1 (0–3)	ns
Vascular risk factors				
Hypertension, *n* (%)	98 (78%)	33 (76%)	65 (78%)	ns
Atrial fibrillation, *n* (%)	36 (29%)	16 (37%)	20 (24%)	ns
Diabetes, *n* (%)	29 (23%)	8 (19%)	21 (25%)	ns
Smoking, *n* (%)	54 (43%)	20 (47%)	42 (50%)	ns
BMI class, *n* (%)				
Underweight	2 (2%)	0	2 (2%)	ns
Normal	43 (34%)	17 (39%)	26 (31%)	
Overweight	47 (37%)	14 (32%)	33 (40%)	
Obese	20 (16%)	5 (12%)	15 (18%)	
Hypercholesterolemia, *n* (%)	83 (66%)	29 (67%)	56 (67%)	ns
Hypertriglyceridemia, *n* (%)	7 (6%)	3 (7%)	8 (10%)	ns
NIHSS, median (IQR)	7 (4–14)	4 (3–5)	10 (6.5–17.5)	***p* < 0.001**
MoCA-B, median (IQR)	23 (18–26)	24.5 (20–27)	21 (17.3–26)	ns
CDT, median (IQR)	9 (7–11)	9 (7.5–12)	9 (6–11)	ns
Thrombolysis, *n* (%)	28 (22%)	4 (9%)	24 (29%)	***p* < 0.05**
Thrombectomy, *n* (%)	16 (13%)	1 (2%)	15 (18%)	***p* < 0.05**
Imaging modality, *n* (%)				
CT	56 (44%)	24 (56%)	34 (41%)	ns
MRI	2 (2%)	0	2 (2%)	
Both	68 (54%)	19 (44%)	46 (55%)	
Infarct laterality, *n* (%)				
Right	42 (33%)	15 (35%)	27 (32%)	ns
Left	72 (57%)	22 (51%)	50 (60%)	
Bilateral	12 (10%)	6 (14%)	6 (7%)	
Infarct location, *n* (%)				
Cortical	74 (59%)	24 (56%)	51 (61%)	ns
Subcortical	46 (36%)	14 (33%)	25 (30%)	
Cerebellar	4 (3%)	3 (7%)	4 (5%)	
Brainstem	2 (2%)	2 (4%)	3 (4%)	
Arterial territory, *n* (%)				
ACA	1 (1%)	0	1 (1%)	ns
MCA	111 (88%)	39 (90%)	72 (87%)	
PCA	8 (6%)	2 (5%)	6 (7%)	
VB	6 (5%)	2 (5%)	4 (5%)	

TABLE 2 (Continued)

	Dysphagic at admission, *N* = 126 (2 deaths)	Non-dysphagic at discharge, *N* = 43 (34%)	Dysphagic at discharge, *N* = 83 (66%)	Univariate analysis
Oxfordshire class, *n* (%)				
LACI	23 (18%)	9 (21%)	14 (17%)	***p* < 0.05**
PACI	71 (56%)	31 (72%)	40 (48%)	
POCI	9 (7%)	3 (7%)	6 (7%)	
TACI	23 (18%)	0	23 (28%)	
Frontal operculum involvement, *n* (%)				
Opercular lesion	53 (42%)	4 (9%)	49 (59%)	***p* < 0.001**
Right opercular	19	1 (2%)	18 (22%)	
Left opercular	34	3 (7%)	27 (32%)	
White matter lesions				
van Swieten scale, median (IQR)	2 (1–4)	2 (1–4)	2 (1–4)	ns
Fazekas scale, median (IQR)	2 (1–4)	2 (1–3)	3 (1–5)	ns
Cortical atrophy				
Global Cortical Atrophy Scale, median (IQR)	14 (8–21)	13.5 (9–18)	14 (8–22)	ns
Scheltens scale, median (IQR)	2 (0–2)	0 (0–2)	2 (0–2)	ns
Stroke etiology, *n* (%)				
LAA	34 (27%)	6 (14%)	20 (24%)	ns
SVO	30 (24%)	9 (21%)	18 (22%)	
CE	35 (28%)	17 (39%)	22 (26%)	
Other	1 (1%)	0	1 (1%)	
Undetermined	21 (17%)	7 (16%)	19 (23%)	
Multifactorial	5 (4%)	4 (9%)	3 (4%)	
Swallowing tests				
FOIS, median (IQR)	4 (4–6)	6 (6–6)	4 (1–4)	***p* < 0.05**
Stroke outcome at discharge				
mRS, median (IQR)	3 (1–4)	1 (1–3)	4 (2–5)	***p* < 0.001**
NIHSS, median (IQR)	3 (1–7)	2 (0.5–3)	5 (2.5–9.5)	***p* < 0.001**
Barthel, median (IQR)	60 (25–100)	85 (62.5–100)	40 (10–75)	***p* < 0.001**
Length of hospital stay, days, median (IQR)	10 (8–15)	9 (7–11.5)	12 (8–17.5)	***p* < 0.05**
Discharge (destination)				
Home	43 (34%)	25 (58%)	18 (21%)	***p* < 0.001**
Rehabilitation	56 (44%)	14 (41%)	48 (58%)	
Nursing home	27 (21%)	4 (5%)	17 (20%)	
Death	2 (0.1%)	0	2 (2%)	

Abbreviations: ACA, anterior cerebral artery; ADL, activities of daily living; BMI, body mass index; CDT, Clock Drawing Test; CE, cardioembolic; CDR, Clinical Dementia Rating scale; CT, computed tomography; FOIS, Functional Oral Intake Scale; IADL, instrumental activities of daily living; IQR, interquartile range; LAA, large artery atherosclerosis; LACI, lacunar cerebral infarct; MCA, middle cerebral artery; MoCA-B, Montreal Cognitive Assessment—Basic; MRI, magnetic resonance imaging; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; NPI, Neuropsychiatric Inventory; PACI, partial anterior cerebral infarct; PCA, posterior cerebral artery; POCI, posterior cerebral infarct; SVO, small vessel occlusion; TACI, total anterior cerebral infarct; VB, vertebrobasilar.

dysphagia score" (PreDyScore) [42] and the "Predictive Swallowing Score" (PRESS) [31].

Our study confirms the role of some previously highlighted predictive factors of post-stroke dysphagia, like the ischaemic lesion extension, frontal operculum involvement, the patient's age and pre-stroke functional status (mRS). Differently from the two previous studies, our study highlights the importance of thrombolysis as a protective factor against dysphagia, stressing once again the pivotal role of recombinant tissue plasminogen activator treatment in acute stroke management. Furthermore, education was a protective factor

TABLE 3 Demographic, clinical and neuroimaging characteristics of patients according to the presence/absence of dysphagia at stroke unit discharge.

	All, $N = 226$	Non-dysphagic, $N = 144$ (64%)	Dysphagic, $N = 82$ (36%)	Univariate analysis
Sex, *n* (%)				
Female	109 (49%)	65 (45%)	44 (54%)	ns
Male	117 (51%)	79 (55%)	38 (46%)	
Age, years, mean (SD)	76.3 (11.9)	74.2 (12.9)	78.9 (10.4)	$p < 0.05$
Education, years, median (IQR)	8 (5–11)	8 (5–12)	6 (5–8)	$p < 0.001$
mRS pre-event, median (IQR)	0 (0–1)	0 (0–1)	0 (0–2)	$p < 0.05$
Lost ADL, median (IQR)	0 (0–1)	0 (0–0)	1 (0–1)	ns
Lost IADL, median (IQR)	0 (0–1)	0 (0–1)	0 (0–2)	ns
CDR, median (IQR)	0 (0–0.5)	0 (0–0)	0 (0–0.5)	ns
NPI, median (IQR)	1 (0–3)	1 (0–3)	1 (0–3)	ns
Vascular risk factors				
Hypertension, *n* (%)	181 (80%)	117 (81%)	64 (78%)	ns
Atrial fibrillation, *n* (%)	66 (29%)	46 (32%)	20 (24%)	ns
Diabetes	53 (23%)	33 (23%)	20 (24%)	ns
Smoke, *n* (%)	106 (47%)	69 (48%)	37 (45%)	ns
BMI class, *n* (%)				
Underweight	4 (1%)	2 (1%)	2 (2%)	Normal vs. all: $p < 0.05$
Normal	95 (42%)	69 (48%)	26 (32%)	
Overweight	86 (38%)	54 (37%)	32 (39%)	
Obese	41 (18%)	19 (13%)	22 (27%)	
Hypercholesterolemia, *n* (%)	146 (65%)	92 (64%)	54 (66%)	ns
Hypertriglyceridemia, *n* (%)	11 (5%)	6 (4%)	5 (6%)	ns
NIHSS, median (IQR)	4 (2–8)	3 (1–4)	10 (6–18)	$p < 0.001$
MoCA-B, median (IQR)	23 (18–26)	23 (18–26)	21 (17–26)	ns
CDT, median (IQR)	9 (6–11)	9 (6–12)	9 (6–11)	ns
Thrombolysis, *n* (%)	44 (19.5%)	20 (14%)	24 (29%)	$p < 0.01$
Thrombectomy, *n* (%)	23 (10.2%)	8 (6%)	15 (18%)	ns
Imaging modality, *n* (%)				
CT	91 (40%)	57 (40%)	34 (41%)	ns
MRI	4 (2%)	2 (1%)	2 (3%)	
Both	131 (58%)	85 (59%)	46 (56%)	
Infarct laterality				
Right	81 (36%)	55 (38%)	26 (32%)	ns
Left	129 (57%)	79 (55%)	50 (61%)	
Bilateral	16 (7%)	10 (7%)	6 (7%)	
Infarct location				
Cortical	121 (53%)	67 (46%)	54 (67%)	$p < 0.01$
Subcortical	89 (39%)	70 (49%)	19 (23%)	
Cerebellar	10 (4%)	4 (3%)	6 (7%)	
Brainstem	6 (3%)	3 (2%)	3 (3%)	
Arterial territory				
ACA	2 (1%)	1 (0,7%)	1 (1%)	ns
MCA	194 (86%)	123 (85%)	71 (87%)	
PCA	14 (6%)	8 (6%)	6 (7%)	
VB	16 (7%)	12 (8%)	4 (5%)	

TABLE 3 (Continued)

	All, $N = 226$	Non-dysphagic, $N = 144$ (64%)	Dysphagic, $N = 82$ (36%)	Univariate analysis
Oxford class				
LACI	41 (18%)	27 (19%)	14 (17%)	TACI vs. all: $p < 0.001$ PACI vs. all: $p < 0.01$
PACI	136 (60%)	97 (67%)	39 (48%)	
POCI	25 (11%)	19 (13%)	6 (7%)	
TACI	24 (11%)	1 (0.7%)	23 (28%)	
Frontal operculum involvement				
Opercular lesion, *n* (%)	60 (26%)	12 (8%)	48 (58%)	$p < 0.001$
Right opercular	20 (9%)			
Left opercular	40 (18%)			
White matter lesion				
van Swieten scale, median (IQR)	2 (1–4)	2 (1–4)	2 (1–4)	ns
Fazekas scale, median (IQR)	3 (1–5)	2 (1–4)	3 (1–4)	ns
Cortical atrophy				
Global Cortical Atrophy Scale, median (IQR)	15 (8–20)	15 (9–20)	14 (8–22)	ns
Scheltens scale, median (IQR)	2 (0–2)	2 (0–2)	2 (0–2)	ns
Stroke etiology				
LAA	58 (26%)	37 (26%)	21 (26%)	ns
SVO	55 (24%)	36 (25%)	19 (23%)	
CE	62 (27%)	37 (26%)	25 (30%)	
Other	4 (2%)	3 (2%)	1 (1%)	
Undetermined	40 (18%)	26 (18%)	14 (17%)	
Multifactorial	7 (3%)	5 (3%)	2 (2%)	
Swallowing tests				
FOIS, median (IQR)	6 (5–7)	7 (6–7)	4 (1–4)	$p < 0.001$
Stroke outcome at discharge				
mRS, median (IQR)	1 (0–4)	1 (0–2)	4 (2–5)	$p < 0.001$
NIHSS, median (IQR)	2 (0–4)	1 (0–2)	5 (2–9)	$p < 0.001$
Barthel, median (IQR)	90 (50–100)	100 (85–100)	40 (10–75)	$p < 0.001$
Length of hospital stay, days, median (IQR)	9 (6–13)	8 (6–10)	12 (8–18)	$p < 0.001$
Discharge (destination)				
Home	129 (57%)	111 (77%)	18 (21%)	$p < 0.001$
Rehabilitation	68 (30%)	26 (18%)	42 (50%)	
Nursing home	29 (12%)	7 (5%)	22 (26%)	
Death	2 (1%)	0 (0%)	2 (2%)	

Abbreviations: ACA, anterior cerebral artery; ADL, activities of daily living; BMI, body mass index; CDT, Clock Drawing Test; CE, cardioembolic; CDR, Clinical Dementia Rating scale; CT, computed tomography; FOIS, Functional Oral Intake Scale; IADL, instrumental activities of daily living; IQR, interquartile range; LAA, large artery atherosclerosis; LACI, lacunar cerebral infarct; MCA, middle cerebral artery; MoCA-B, Montreal Cognitive Assessment—Basic; MRI, magnetic resonance imaging; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; NPI, Neuropsychiatric Inventory; PACI, partial anterior cerebral infarct; PCA, posterior cerebral artery; POCI, posterior cerebral infarct; SVO, small vessel occlusion; TACI, total anterior cerebral infarct; VB, vertebrobasilar.

for dysphagia and its persistence. At present, these data remain unexplained also considering that cognitive scores were not protective. The latter result has the obvious caveat that our cognitive measures were limited to two brief tests. Nevertheless, these data are of interest because they imply that cognitive status does not predict dysphagia in the acute phase and no pre-selection of patients for dysphagia screening should be done, and therefore all stroke patients need to be evaluated for dysphagia. It is instead known that cognitive status interferes with dysphagia recovery in the chronic stages [48, 49].

TABLE 4 NOTTEM (NIHSS, operculum, TACI, thrombolysis, education, mRS) score.

Item	Values	Points
NIHSS at admission	<4	0
	4 ≤ NIHSS ≤ 7	1
	>7	2
Operculum	Absence of frontal opercular lesion	0
	Presence of frontal opercular lesion	2
TACI	No	0
	Yes	2
Thrombolysis	No	1
	Yes	0
Education	Years of school ≤7	1
	Years of school >7	0
mRS at admission	<2	0
	≥2	1
NOTTEM score		Total:___

Abbreviations: mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; TACI, total anterior circulation infarct.

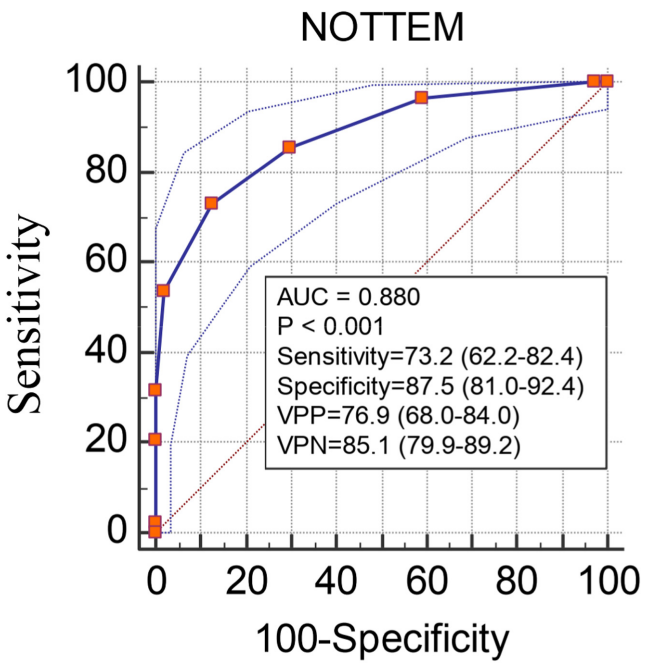

FIGURE 1 ROC curve for NOTTEM score >3.

TABLE 5 Sensitivity, specificity, positive and negative predictive values for NOTTEM score.

Criterion	Sensitivity (95% CI)	Specificity (95% CI)	Positive predictive value (95% CI)	Negative predictive value (95% CI)
≥0	100.00 (95.6–100.0)	0.00 (0.0–2.5)	36.3 (36.3–36.3)	
>0	100.00 (95.6–100.0)	2.78 (0.8–7.0)	36.9 (36.3–37.6)	100.0
>1	96.34 (89.7–99.2)	40.97 (32.9–49.5)	48.2 (44.6–51.7)	95.2 (86.4–98.4)
>2	85.37 (75.8–92.2)	70.14 (62.0–77.5)	61.9 (55.5–68.0)	89.4 (83.2–93.5)
>3	73.17 (62.2–82.4)	87.50 (81.0–92.4)	76.9 (68.0–84.0)	85.1 (79.9–89.2)
>4	53.66 (42.3–64.7)	97.92 (94.0–99.6)	93.6 (82.5–97.9)	78.8 (74.6–82.4)
>5	31.71 (21.9–42.9)	100.00 (97.5–100.0)	100.0	72.0 (68.9–74.9)
>6	20.73 (12.6–31.1)	100.00 (97.5–100.0)	100.0	68.9 (66.5–71.2)
>7	2.44 (0.3–8.5)	100.00 (97.5–100.0)	100.0	64.3 (63.5–65.1)
>8	0.00 (0.0–4.4)	100.00 (97.5–100.0)		63.7 (63.7–63.7)

Another difference from the PRESS study was that the population of our study included less severe stroke patients (median baseline NIHSS of 12 in the PRESS study vs. median NIHSS 4 in our study) [31].

This study has several limitations. First is the retrospective nature. Furthermore, the results are applicable only to patients with ischaemic stroke, as this was the focus of our analyses. Moreover, the heterogeneity in the imaging modalities (CT or MRI) could have affected the detection of lesions in strategic sites (e.g., small opercular lesions are not easily detectable in CT scans) in some patients. Moreover, in this study, the presence of dysphagia was defined as an FOIS score ≤6. This cut-off was chosen arbitrarily, to maximize sensitivity for dysphagia. In previous studies assessing FOIS validity against a videofluoroscopic swallowing study and FEES, a linear correlation between the methods was underlined, but a specific cut-off for dysphagia diagnosis was not defined. Only a small minority of our patients underwent FEES. Therefore, the presence of laryngopharyngeal reflux as a cause of aspiration cannot be excluded in all patients. Finally, the NOTTEM score needs future validation in an independent sample.

The strength of the study is the creation of a clinical prognostic score with good diagnostic accuracy and easy applicability in everyday practice by medical staff, constituting important decisional support in the implementation of nutritional invasive strategies like NGT or PEG placement.

CONCLUSIONS

In conclusion, predictors of dysphagia were found in acute stroke patients and a score that might be useful was proposed, evaluating clinical and neuroimaging variables at admission, to estimate the risk of dysphagia persistence during the first 2 weeks of stroke unit stay. It was not possible to document an effect of cognitive measures on dysphagia in the acute stroke phase. The role of cognition on dysphagia recovery remains to be assessed. Moreover, a validation of the NOTTEM score in an independent sample needs to be assessed as a future perspective.

AUTHOR CONTRIBUTIONS

Daniele Mattavelli: Conceptualization; investigation; writing – original draft; methodology; validation; software; formal analysis; data curation; supervision; project administration; visualization. **Francesco Mele:** Data curation; validation; visualization; supervision; methodology. **Ilaria Cova:** Validation; methodology; visualization; data curation; supervision. **Silvia Rosa:** Validation; visualization; supervision. **Pierluigi Bertora:** Validation; visualization; supervision. **Simone Pomati:** Methodology; validation; visualization; supervision. **Nicole Pizzorni:** Methodology; validation; visualization; supervision. **Antonio Schindler:** Methodology; validation; visualization; supervision. **Leonardo Pantoni:** Conceptualization; methodology; validation; visualization; project administration; data curation; supervision; resources; funding acquisition.

FUNDING INFORMATION

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CONFLICT OF INTEREST STATEMENT

Daniele Mattavelli, Francesco Mele, Ilaria Cova, Silvia Rosa, Pierluigi Bertora, Simone Pomati, Nicole Pizzorni, Antonio Schindler declare that there is no conflict of interest. Leonardo Pantoni is a member of the editorial boards of *Neurology*, *Stroke*, *European Stroke Journal*, *Cerebrovascular Diseases*, *Cerebral Circulation Cognition and Behavior*, and associate editor of *Neurological Sciences*. He reports no other disclosure.

DATA AVAILABILITY STATEMENT

Data that support the findings of this study are available on request from the corresponding author. Data are not publicly available due to privacy or ethical restrictions.

ETHICAL APPROVAL AND INFORMED CONSENT

Ethical approval was not sought for the present study because this study was retrospective and all data were gathered as part of routine hospital care for which written consensus is collected for each patient or caregiver. All the procedures were carried out in accordance with the Declaration of Helsinki.

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REFERENCES

1. Martino R, Foley N, Bhogal S, Diamant N, Speechley M, Teasell R. Dysphagia after stroke: incidence, diagnosis, and pulmonary complications. *Stroke*. 2005;36(12):2756-2763. doi:10.1161/01.STR.0000190056.76543.eb
2. Takizawa C, Gemmell E, Kenworthy J, Speyer R. A systematic review of the prevalence of oropharyngeal dysphagia in stroke, Parkinson's disease, Alzheimer's disease, head injury, and pneumonia. *Dysphagia*. 2016;31(3):434-441. doi:10.1007/s00455-016-9695-9
3. D'Netto P, Rumbach A, Dunn K, Finch E. Clinical predictors of dysphagia recovery after stroke: a systematic review. *Dysphagia*. 2023 Feb;38(1):1-22. doi:10.1007/s00455-022-10443-3
4. Dziewas R, Michou E, Trapl-Grundschober M, et al. European Stroke Organisation and European Society for Swallowing Disorders guideline for the diagnosis and treatment of post-stroke dysphagia. *Eur Stroke J*. 2021;6(3):LXXXIX-LXXCXV. doi:10.1177/23969873211039721
5. Nascimento A, Carvalho M, Nogueira J, Abreu P, Nzwalo H. Complications associated with nasogastric tube placement in the acute phase of stroke: a systematic review. *J Neurosci Nurs*. 2018;50(4):193-198. doi:10.1097/JNN.0000000000000372
6. Aslanyan S, Weir CJ, Diener HC, Kaste M, Lees KR, GAIN International Steering Committee and Investigators. Pneumonia and urinary tract infection after acute ischaemic stroke: a tertiary analysis of the GAIN international trial. *Eur J Neurol*. 2004;11(1):49-53. doi:10.1046/j.1468-1331.2003.00749.x
7. Perry L, Love CP. Screening for dysphagia and aspiration in acute stroke: a systematic review. *Dysphagia*. 2001;16(1):7-18. doi:10.1007/pl00021290
8. Falsetti P, Acciai C, Palilla R, 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):329-335. doi:10.1016/j.jstrokecerebrovasdis.2009.01.009
9. Holas MA, DePippo KL, Reding MJ. Aspiration and relative risk of medical complications following stroke. *Arch Neurol*. 1994;51(10):1051-1053. doi:10.1001/archneur.1994.00540220099020
10. FOOD Trial Collaboration. Poor nutritional status on admission predicts poor outcomes after stroke: observational data from the FOOD trial. *Stroke*. 2003;34(6):1450-1456. doi:10.1161/01.STR.0000074037.49197.8C
11. Hurford R, Charidimou A, Fox Z, Cipolotti L, Werring DJ. Domain-specific trends in cognitive impairment after acute ischaemic stroke. *J Neurol*. 2013;260(1):237-241. doi:10.1007/s00415-012-6625-0
12. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. *Hypertension*. 2018;71(6):e13-e115. doi:10.1161/HYP.0000000000000065
13. Kirchhof P, Benussi S, Kotecha D, et al. 2016 ESC guidelines for the management of atrial fibrillation developed in collaboration with EACTS. *Eur Heart J*. 2016;37(38):2893-2962. doi:10.1093/eurheartj/ehw210
14. Deckers JG, Schellevis FG, Fleming DM. WHO diagnostic criteria as a validation tool for the diagnosis of diabetes mellitus: a study in five European countries. *Eur J Gen Pract*. 2006;12(3):108-113. doi:10.1080/13814780600881268

15. Mach F, Baigent C, Catapano AL, et al. 2019 ESC/EAS guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. *Eur Heart J*. 2020;41(1):111-188. doi:10.1093/eurheartj/ehz455
16. National Institute of Neurological Disorders and Stroke. Rt-PA stroke study group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med*. 1995;333(24):1581-1587. doi:10.1056/NEJM199512143332401
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):1521-1526. doi:10.1016/0140-6736(91)93206-o
18. Julayanont P, Tangwongchai S, Hemrungrojn S, et al. The Montreal Cognitive Assessment—Basic: a screening tool for mild cognitive impairment in illiterate and low-educated elderly adults. *J Am Geriatr Soc*. 2015;63(12):2550-2554. doi:10.1111/jgs.13820
19. Caffarra P, Gardini S, Zonato F, et al. Italian norms for the Freedman version of the clock drawing test. *J Clin Exp Neuropsychol*. 2011;33(9):982-988. doi:10.1080/13803395.2011.589373
20. Hughes CP, Berg L, Danziger WL, Coben LA, Martin RL. A new clinical scale for the staging of dementia. *Br J Psychiatry*. 1982;140:566-572. doi:10.1192/bjp.140.6.566
21. Cummings JL. The neuropsychiatric inventory: assessing psychopathology in dementia patients. *Neurology*. 1997;48(5 Suppl 6):S10-S16. doi:10.1212/wnl.48.5_suppl_6.10s
22. Katz S, Downs TD, Cash HR, Grotz RC. Progress in development of the index of ADL. *Gerontologist*. 1970;10(1):20-30. doi:10.1093/geront/10.1_part_1.20
23. Lawton MP, Brody EM. Assessment of older people: self-maintaining and instrumental activities of daily living. *Gerontologist*. 1969;9(3):179-186.
24. Chung JW, Park SH, Kim N, et al. Trial of ORG 10172 in acute stroke treatment (TOAST) classification and vascular territory of ischemic stroke lesions diagnosed by diffusion-weighted imaging. *J Am Heart Assoc*. 2014;3(4):e001119. doi:10.1161/JAHA.114.001119
25. van Swieten JC, Koudstaal PJ, Visser MC, Schouten HJ, van Gijn J. Interobserver agreement for the assessment of handicap in stroke patients. *Stroke*. 1988;19(5):604-607. doi:10.1161/01.str.19.5.604
26. Mahoney FI, Barthel DW. Functional evaluation: the Barthel index. *Md State Med J*. 1965;14:61-65.
27. Martino R, Silver F, Teasell R, et al. The Toronto bedside swallowing screening test (TOR-BSST): development and validation of a dysphagia screening tool for patients with stroke. *Stroke*. 2009;40(2):555-561. doi:10.1161/STROKEAHA.107.510370
28. Crary MA, Mann GD, Groher ME. Initial psychometric assessment of a functional oral intake scale for dysphagia in stroke patients. *Arch Phys Med Rehabil*. 2005;86(8):1516-1520. doi:10.1016/j.apmr.2004.11.049
29. Battel I, Calvo I, Walshe M. Cross-cultural validation of the Italian version of the Functional Oral Intake Scale. *Folia Phoniatr Logop*. 2018;70(3–4):117-123. doi:10.1159/000490792
30. Ninfa A, Pizzorni N, Eplite A, Moltisanti C, Schindler A. Validation of the Italian version of the Functional Oral Intake Scale (FOIS-it) against fiberoptic endoscopic evaluation of swallowing and nutritional status. *Dysphagia*. 2022;37(1):137-147. doi:10.1007/s00455-021-10257-9
31. Galovic M, Stauber AJ, Leisi N, et al. Development and validation of a prognostic model of swallowing recovery and enteral tube feeding after ischemic stroke. *JAMA Neurol*. 2019;76(5):561-570. doi:10.1001/jamaneurol.2018.4858
32. van Swieten JC, Hijdra A, Koudstaal PJ, van Gijn J. Grading white matter lesions on CT and MRI: a simple scale. *J Neurol Neurosurg Psychiatry*. 1990;53(12):1080-1083. doi:10.1136/jnnp.53.12.1080
33. Fazekas F, Chawluk JB, Alavi A, Hurtig HI, Zimmerman RA. MR signal abnormalities at 1.5 T in Alzheimer's dementia and normal aging. *AJR Am J Roentgenol*. 1987;149(2):351-356. doi:10.2214/ajr.149.2.351
34. Pasquier F, Leys D, Weerts JG, Mounier-Vehier F, Barkhof F, Scheltens P. Inter- and intraobserver reproducibility of cerebral atrophy assessment on MRI scans with hemispheric infarcts. *Eur Neurol*. 1996;36(5):268-272. doi:10.1159/000117270
35. Scheltens P, Launer LJ, Barkhof F, Weinstein HC, van Gool WA. Visual assessment of medial temporal lobe atrophy on magnetic resonance imaging: interobserver reliability. *J Neurol*. 1995;242(9):557-560. doi:10.1007/BF00868807
36. Rofes L, Muriana D, Palomeras E, et al. Prevalence, risk factors and complications of oropharyngeal dysphagia in stroke patients: a cohort study. *Neurogastroenterol Motil*. 2018;23:e13338. doi:10.1111/nmo.13338
37. Mann G, Hankey GJ. Initial clinical and demographic predictors of swallowing impairment following acute stroke. *Dysphagia*. 2001;16(3):208-215. doi:10.1007/s00455-001-0069-5
38. Broadley S, Croser D, Cottrell J, et al. Predictors of prolonged dysphagia following acute stroke. *J Clin Neurosci*. 2003;10(3):300-305. doi:10.1016/s0967-5868(03)00022-5
39. Lapa S, Foerch C, Singer OC, Hattingen E, Luger S. Ischemic lesion location based on the ASPECT score for risk assessment of neurogenic dysphagia. *Dysphagia*. 2021;36(5):882-890. doi:10.1007/s00455-020-10204-0
40. Lin WC, Huang CY, Lee LF, Chen YW, Ho CH, Sun YT. Initial National Institutes of Health Stroke Scale to early predict the improvement of swallowing in patients with acute ischemic stroke. *J Stroke Cerebrovasc Dis*. 2019;28(10):104297. doi:10.1016/j.jstrokecerebrovasdis.2019.07.013
41. Beharry A, Michel P, Faouzi M, Kuntzer T, Schweizer V, Diserens K. Predictive factors of swallowing disorders and bronchopneumonia in acute ischemic stroke. *J Stroke Cerebrovasc Dis*. 2019;28(8):2148-2154. doi:10.1016/j.jstrokecerebrovasdis.2019.04.025
42. Gandolfo C, Sukkar S, Ceravolo MG, et al. The predictive dysphagia score (PreDyScore) in the short- and medium-term post-stroke: a putative tool in PEG indication. *Neurol Sci*. 2019;40(8):1619-1626. doi:10.1007/s10072-019-03896-2
43. Brown K, Cai C, Barreto A, et al. Predictors of percutaneous endoscopic gastrostomy placement in acute ischemic stroke. *J Stroke Cerebrovasc Dis*. 2018;27(11):3200-3207. doi:10.1016/j.jstrokecerebrovasdis.2018.07.022
44. Westendorp WF, Vermeij JD, Hilkens NA, et al. Development and internal validation of a prediction rule for post-stroke infection and post-stroke pneumonia in acute stroke patients. *Eur Stroke J*. 2018;3(2):136-144. doi:10.1177/2396987318764519
45. Labeit B, Mueller H, Muhle P, et al. Predicting dysphagia with National Institutes of Health Stroke Scale: distinction between infra- and supratentorial region is essential. *Cerebrovasc Dis*. 2018;46(3–4):152-160. doi:10.1159/000493371
46. Jeyaseelan RD, Vargo MM, Chae J. National Institutes of Health Stroke Scale (NIHSS) as an early predictor of poststroke dysphagia. *PM R*. 2015;7(6):593-598. doi:10.1016/j.pmrj.2014.12.007
47. Daniels SK, Pathak S, Mukhi SV, Stach CB, Morgan RO, Anderson JA. The relationship between lesion localization and dysphagia in acute stroke. *Dysphagia*. 2017;32(6):777-784. doi:10.1007/s00455-017-9824-0
48. Jo SY, Hwang JW, Pyun SB. Relationship between cognitive function and dysphagia after stroke. *Ann Rehabil Med*. 2017;41(4):564-572. doi:10.5535/arm.2017.41.4.564
49. Calvo I, Pizzorni N, Gilardone G, et al. Predictors of oral feeding resumption after stroke in a rehabilitation hospital: a retrospective

study. *J Stroke Cerebrovasc Dis*. 2019;28(7):1958-1970. doi:10.1016/j.jstrokecerebrovasdis.2019.03.040

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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