Prevalence, risk factors and complications of oropharyngeal dysphagia in stroke patients: A cohort study

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Abstract

Background: Oropharyngeal dysphagia (OD) is a prevalent poststroke condition with severe complications and increased mortality. Poststroke OD prevalence varies among studies and there is little evidence of its related risk factors and associated complications. Objective: to evaluate the prevalence of OD after stroke and the risk factors and associated complications.

Methods: We performed a prospective longitudinal study of stroke patients consecutively admitted to a general hospital. OD was diagnosed with the volume-viscosity swallow test (V-VST). Demographic, functional status and topographical and clinical variables of stroke were collected to assess risk factors for OD. We evaluated functional status, mortality, respiratory infections, and readmissions 3 and 12 months after stroke. A multivariate regression analysis determined associated risk factors for OD and for each outcome variable.

Key Results: We included 395 stroke patients with a 45.06% prevalence of OD on admission. OD was independently associated with age (OR = 1.05; CI = 1.02-1.08), previous stroke (OR = 2.40; CI = 1.00-5.79), severity using the National Institute of Health Stroke Scale (OR = 3.52; CI = 1.57-7.87) and volume of the lesion (OR = 1.02; CI = 1.01-1.03). OD after stroke was an independent risk factor for prolonged hospital stay (P = .049; β = 0.938) and institutionalization after discharge (OR = 0.47; CI = 0.24-0.92); OD was an independent risk factor for poorer functional capacity (OR = 3.00; CI = 1.58-5.68) and increased mortality (HR = 6.90; CI = 1.57-30.34) 3 months after stroke.

Conclusions & Inferences: Poststroke OD is prevalent and associated with poor short and long term prognosis. Stroke severity and patient status before stroke were more relevant to OD than lesion location. Systematic screening programs and early OD management could significantly improve poststroke patient outcome.

Key Words
cohort study, dysphagia, prevalence studies, risk factors, stroke
1 | INTRODUCTION

Oropharyngeal dysphagia (OD) is a common morbidity after stroke that impairs swallowing physiology. Poststroke OD is a condition recognized by the World Health Organization with the following International Classification of Diseases (ICD) codes: 438.82 in the ICD-9 and I69.391 in the ICD-10.1 OD after stroke has been associated with complications such as malnutrition and respiratory infections which lead to poor prognosis and high mortality rates when compared with stroke without dysphagia.2-5

Poststroke dysphagia remains a neglected area of research despite its high impact on prognosis.6 An indication of this neglect is that the prevalence of OD following stroke varies from 19% to 81%5 depending on study methodology and inclusion criteria. In a systematic review, Martino et al.5 encouraged further research, not only to assess the real prevalence of OD after stroke, but also to examine the influence of other variables such as age, stroke type or stroke severity. Few studies have evaluated the risk factors for OD after stroke,2-12 and even fewer used appropriate study design to determine their relationship.4,7,9 Some studies have focused on sociodemographic and/or clinical factors, and others have mainly analyzed topographical factors. To date, older age, state of consciousness, and severe neurological deficits on admission are the most commonly reported risk factors, but not all studies report the same factors.7-12 Moreover, severity of stroke has been studied in many different ways and the relationship between neurotopography and OD after stroke is still controversial.7,9,11,13-16

A clear picture of the true prevalence of stroke-associated OD, the main risk factors and its effects on prognosis will improve stroke patient management. Our study had three main aims: first, to determine the prevalence of OD in acute stroke patients admitted to a general hospital; secondly, to establish sociodemographic and clinical risk factors for poststroke OD, and thirdly, to determine the impact of OD on the clinical outcome and 1-year prognosis of the patient.

2 | METHODS

2.1 | Study design and patient characteristics

An observational prospective cohort study of stroke patients consecutively admitted to a general hospital, from May 2010 to September 2014, was performed.

Patients admitted to the hospital with clinical suspicion of stroke were invited to participate in the study. Only those patients with confirmed stroke diagnosis continued in the study. Exclusion criteria were previous diagnosis of OD (to ensure that OD was caused by the present stroke), transient ischemic attack (due to the quick reversibility of symptoms), and being transferred from another hospital (to keep track of the patients from the very acute phase).

2.2 | Sample size

To determine the optimum sample size for the study, a power analysis was performed and found that a sample size of 384 people will suffice to estimate with a 95% confidence and a precision ± 5 percent units, a population prevalence considered to be around 51% (OD prevalence assessed by clinical methods).5

2.3 | Standard protocol approvals, registrations, and patient consents

The study protocol was approved by the ethical committee of the Hospital de Mataró (protocol code 17/11) and registered in the ClinicalTrials.Gov (Code NCT03147755). All procedures were conducted according to the principles and rules laid down in the Declaration of Helsinki and its subsequent amendments and following the current Spanish laws for data protection (LO 15/1999). All patients included in the study (or a legal representative) signed an informed consent.

2.4 | Data collected

2.4.1 | Oropharyngeal dysphagia diagnosis

Oropharyngeal dysphagia was clinically assessed by especially trained nursing staff, using a validated swallowing test, the volume viscosity swallow test (V-VST).17,18 The V-VST is a clinical assessment
method developed by our group that uses swallow boluses of different volumes (5, 10 and 20 mL) and viscosities (thin liquid, nectar-like and spoon thick). The test allows swallow safety and efficacy to be assessed with the minimum risk for the patient, following the methodology previously described.17,18

1. Signs and symptoms of impaired efficacy of swallow: the signs of impaired efficacy of swallow directly evaluated for each swallow were the presence of oral residue (part of the bolus remaining in the oral cavity after swallow), the efficiency of labial seal (ability to maintain the whole bolus in the oral cavity during the preparatory phase of swallow) and fractional swallow (multiple swallows per bolus); the presence of pharyngeal residue was detected by asking patients if something felt stuck or remained in the throat after each swallow.

2. Signs of impaired safety of swallow: the signs of impaired safety of swallow were detected for each swallow through changes in voice quality (including wet voice), cough and decrease in oxygen saturation ≥3% from the basal level (measured with a finger pulse-oximeter (Nellcor OxiMax, Philips Medical Systems, Eindhoven, The Netherlands) to detect silent aspirations.

This clinical test has been validated in several phenotypes of patients with OD including patients with poststroke OD, showing excellent psychometric characteristics,18 0.94 sensitivity and 0.88 specificity for OD; 0.79 sensitivity and 0.75 specificity for impaired efficacy; 0.87 sensitivity and 0.81 specificity for impaired safety, and 0.91 sensitivity for aspirations.

The V-VST was performed between 24 and 48 hours after admission before oral feeding with the exception of patients with low levels of consciousness. Patients who scored >0 in item 1a(LOC) of NIHSS were maintained in nil per os and considered positive for OD. Signs of impaired safety and efficacy of swallow were evaluated as previously described.17,18 If a patient presented any sign of impaired efficacy and/or safety of swallow, he/she was considered as having OD. We repeated the test on discharge if the admission test had been positive.

2.4.2 | Oropharyngeal dysphagia treatment

Standard treatment of poststroke OD at our institution during this study was mainly compensatory for all patients and included fluid viscosity adaptation19 and texture modified food.20,21 A small subgroup of patients also received individualized swallow therapy provided by an SLP and based on postural changes and manoeuvres.22

2.4.3 | Sociodemographic data

Age, sex, civil status, and abode were collected from all patients.

2.4.4 | Clinical data

Clinical data of the included patients was collected at five time points:

1. Before admission: Medical history of patients was carefully reviewed and completed with patient’s anamnesis. Hypertension, diabetes mellitus, dyslipidemia, heart disease, and previous stroke history was collected for all patients. To assess the status of the patient before the stroke episode, the Charlson Comorbidities Index (CCI)23 and the modified Rankin Scale (mRS)24 were completed with information from medical records and provided by the patient or their relatives.

2. On admission: Neurological status of the patient was assessed according to the National Institute of Health Stroke Scale (NIHSS); for those patients affected by an ischemic stroke, the subtype of the stroke was also assessed according to the Oxford Community Stroke Project Classification.25 For neurotopographic analysis, the cerebral vascular territory in ischemic strokes, laterality of stroke, cortical or subcortical lesion, and supratentorial vs infratentorial characteristics were assessed. The presence of previous lesions was also recorded. Magnetic resonance imaging (MRI) was the test of choice to assess these parameters whenever possible; otherwise, a computerized tomography (CT) scan was performed. Lesion volume was calculated according to the formula (A*B*C)/2.26 Etiology of stroke was determined according to TOAST criteria.27

3. On discharge: The association between OD and the following outcomes was assessed: neurologic complications during hospital stay, such as seizures, stroke recurrence and neurological deterioration defined as increase in NIHSS >4 (>1 in item 1a(LOC) of NIHSS), systemic complications during hospital stay (respiratory and urinary infections), mortality, length of stay and discharge destination. Patients diagnosed with dysphagia on admission were assessed again on discharge, using the V-VST.

4. At 3 months follow-up: V-VST and mRS was assessed in an outpatient clinic visit 3 months after the stroke episode. Patients were interviewed and their electronic medical records were reviewed to assess the occurrence of respiratory infections requiring antibiotic prescription, readmissions to the emergency department or to the hospital wards, institutionalization, and mortality.

5. One-year follow-up: Electronic medical records were reviewed for respiratory infections requiring antibiotic prescription, readmissions to the emergency department, or to the hospital wards, institutionalization, and mortality.

2.5 | Statistical analysis

To determine the proper sample size for the study, a power analysis was performed. A sample size of 384 patients was sufficient to estimate an approximately 51% prevalence of OD with a 95% confidence interval and with a precision of ±5 percent units.5

A descriptive analysis of the main characteristics of the study sample was performed. Categorical variables were described
as absolute and relative frequencies, continuous variables as mean ± SD, and discrete variables as median and ranges.

For the study of association between OD and categorical variables, the chi-squared test or the Fisher exact test were used, and for continuous variables, the Mann-Whitney U test or the t test were used. To determine which variables were independently associated with OD, a multivariate logistic regression analysis was performed.

Likewise, a univariate analysis was used to assess whether OD was associated with the clinical outcomes at discharge, 3 and 12 months follow-up. Multivariate models for each of the clinical outcomes were built using those variables associated with the outcome of interest and OD (for which \( P < .05 \) was determined in the univariate analyses). We used a multivariate logistic regression analysis for the dependent categorical variables, a linear regression analysis for the dependent continuous variables, and a Cox regression analysis to assess the association between OD and mortality.

Odds ratios (OR) (or hazard ratio [HR] for mortality) and 95% confidence intervals (CI) were calculated for those statistically significant variables. Beta coefficient was calculated for the length of stay analysis. A \( P < .05 \) was considered significant in all cases. The SPSS Statistics 15.0 software (SPSS Inc, Chicago, IL, USA) was used to perform the analysis.

3 | RESULTS

3.1 | Demographics and clinical characteristics of the study population

We evaluated 608 patients consecutively admitted to the hospital with clinical suspicion of stroke. Of those, 450 fulfilled inclusion and exclusion criteria and were included in the study. Fifty-five of the included patients were excluded (see Figure S1), so the final sample of patients was 395. The main causes of exclusion were as follows: final diagnosis other than stroke (18) and TIA (22).

Stroke patients included in the study had a mean age of 73.2 ± 13.13 years and 53.4% were men. Table 1 and Table S1 summarize the main sociodemographic and clinical characteristics of the population studied and the main clinical outcomes assessed at discharge, 3 months and 1 year follow-up.

3.2 | Oropharyngeal dysphagia

The prevalence of OD in the studied population on admission was 45.06%. Of those, 5.82% only presented safety impairments, 6.58% only presented efficacy impairments, and 32.66% presented both safety and efficacy impairments. The most prevalent sign of impaired safety of swallow was voice change observed in up to 65.87% patients with OD, and the most prevalent sign of impaired efficacy was fractional swallow observed in up to 65.87% patients with OD. Safety impairment signs were significantly more prevalent in thin liquid series (23.96%) than in higher viscosity series such as nectar (7.08%) and pudding (4.73%) \( P < .001 \).

On discharge, we found that 66.29% of the patients with OD on admission still had OD; 57.87% had signs of impaired safety of swallow and 61.80% had signs of impaired efficacy of swallow. This means that 33.62% of all patients admitted with stroke still had OD on discharge.

3.3 | Risk factors associated with oropharyngeal dysphagia

3.3.1 | Univariate analysis

Older age, female gender, widow civil status, and living in a nursing home were sociodemographic characteristics associated with OD in the univariate analysis (Table 2).

Patients with clinical history of hypertension, heart disease (especially those with major cardioembolic heart disease), and previous stroke (especially cerebral infarcts) were also associated with OD. High CCI and poor functional status before the stroke episode were also significantly associated with OD after stroke.

Finally, stroke clinical factors associated with OD in the univariate analysis were an NIHSS score higher than 6, territorial infarct (vs lacunar), total anterior circulation infarct diagnosis and high volume lesion.

3.3.2 | Multivariate analysis

Older age, previous stroke diagnosis, poor neurological status (NIHSS score >6), and high stroke lesion volume were independent risk factors associated with OD in poststroke patients (Table 3).

3.3.3 | NIHSS items

In the univariate analysis using only NIHSS items as risk factors for OD, we found that all NIHSS items except for ataxia were risk factors for OD. In the multivariate analysis, the level of consciousness (OR = 12.83, \( P < .02 \)), aphasia (OR = 2.00, \( P < .02 \)), dysarthria (OR = 4.03, \( P < .001 \)), and visual field affection (OR = 2.21, \( P < .02 \)) were independent risk factors for OD.

3.4 | Oropharyngeal dysphagia as a prognostic factor after a stroke

3.4.1 | Univariate analysis

During hospital stay, poststroke OD was associated with an increased number of neurological and systemic complications (both respiratory and urinary infections), higher mortality rates, prolonged hospital stay, and increased institutionalization rates following discharge (Table 4). At 3 months follow-up, patients with OD presented a worse functional status and increased respiratory infections and mortality rates than patients without OD, but similar nutritional status and readmission rates. At 12 months follow-up, patients with OD presented higher mortality (Figure 1) and respiratory infection rates than patients without OD.
After adjusting for possible confounding factors, OD remained significantly associated with prolonged hospital stay and higher rates of institutionalization. At 3 months follow-up, poorer functional status and higher mortality were significantly associated with OD. At 1-year follow-up, after adjusting for the respective confounding factors, OD did not remain associated with respiratory infections and mortality.

3.4.2 | Multivariate analysis

Our study showed a 45.06% prevalence of new-onset dysphagia after stroke. We found that older age, previous stroke diagnosis, severity of stroke according to NIHSS and high stroke lesion volume were independent risk factors for OD after stroke. Moreover, we found that presenting OD after stroke was associated with high mortality rates during hospital stay and was an independent risk factor for prolonged length of hospital stay and to be institutionalized after hospital discharge; OD was also an independent risk factor for poorer functional capacity and increased risk of mortality 3 months after the stroke episode.
4.1 | Poststroke OD prevalence

This study included the largest sample size of those that aim to determine OD prevalence and risk factors after stroke, this being one of its major strengths.

The first important result was that the prevalence of OD, evaluated by clinical methods, was 45.06%, which is a bit lower than previously reported results, using similar methods (51-55%5). This difference could be explained by the fact that our population had less severe stroke than other studies as the more severe cases were referred to a tertiary stroke center close to our hospital; there

patients could undergo thrombectomy and invasive treatments according to The Catalan Stroke Code and Reperfusion Consortium,28 as explained in the exclusion criteria of our patients. Another reason why our patients were less severe is because some patients in a critical state were not able to sign the Informed Consent and could not enter the study. This loss of severe stroke patients can be considered a limitation but, if we compare our series with some stroke registries published over recent years, we can see that this sample is quite representative of unselected stroke prospective series.29-32

We repeated the dysphagia test on discharge on those patients that had OD on admission and we found that 66.29% of these

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**TABLE 2** Univariate analysis for oropharyngeal dysphagia (OD) risk factors. Data presented as n (%), except for age, stroke lesion volume and Charlson Index which are presented as mean ± standard deviation. OR and 95% CI (confidence interval) are shown for those variables that were statistically significant associated with OD (P < .05)

<table>
<thead>
<tr>
<th></th>
<th>OD patients (n = 178)</th>
<th>Non-OD patients (n = 217)</th>
<th>P-value</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Socio-demographic characteristics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>77.9 ± 11.1</td>
<td>69.4 ± 13.7</td>
<td>&lt;.001</td>
<td>1.06 (1.04-1.08)</td>
</tr>
<tr>
<td>Sex (women)</td>
<td>93 (52.2%)</td>
<td>91 (41.9%)</td>
<td>.041</td>
<td>1.52 (1.02-2.26)</td>
</tr>
<tr>
<td>Marital status (widowed)</td>
<td>71 (39.9%)</td>
<td>49 (22.6%)</td>
<td>&lt;.001</td>
<td>2.24 (1.45-3.48)</td>
</tr>
<tr>
<td>Living with (nursing home)</td>
<td>17 (9.5%)</td>
<td>3 (1.4%)</td>
<td>&lt;.001</td>
<td>7.53 (2.17-26.14)</td>
</tr>
<tr>
<td><strong>Clinical status before the admission</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>149 (83.7%)</td>
<td>162 (74.7%)</td>
<td>.030</td>
<td>1.74 (1.06-2.88)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>71 (39.9%)</td>
<td>79 (36.4%)</td>
<td>.532</td>
<td>–</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>105 (59.0%)</td>
<td>128 (59.0%)</td>
<td>1.000</td>
<td>–</td>
</tr>
<tr>
<td>Heart disease</td>
<td>61 (34.3%)</td>
<td>49 (22.6%)</td>
<td>.013</td>
<td>1.78 (1.15-2.79)</td>
</tr>
<tr>
<td>Major cardioembolic</td>
<td>42 (23.6%)</td>
<td>29 (13.4%)</td>
<td>.012</td>
<td>2.00 (1.19-3.37)</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>41 (23.0%)</td>
<td>32 (14.7%)</td>
<td>.038</td>
<td>1.73 (1.04-2.89)</td>
</tr>
<tr>
<td>Infarct</td>
<td>28 (15.7%)</td>
<td>17 (7.8%)</td>
<td>.014</td>
<td>2.20 (1.16-4.16)</td>
</tr>
<tr>
<td>Charlson Index</td>
<td>2.9 ± 1.82</td>
<td>2.3 ± 1.67</td>
<td>&lt;.001</td>
<td>1.22 (1.08-1.37)</td>
</tr>
<tr>
<td>mRS (score &gt;1)</td>
<td>64 (36.0%)</td>
<td>33 (15.2%)</td>
<td>&lt;.001</td>
<td>3.13 (1.94-5.06)</td>
</tr>
<tr>
<td><strong>Clinical data on admission</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NIHSS on admission (score &gt;6)</td>
<td>77 (43.3%)</td>
<td>15 (6.9%)</td>
<td>&lt;.001</td>
<td>10.27 (5.62-18.75)</td>
</tr>
<tr>
<td>Neuroradiological findings related to the present stroke (territorial infarction)</td>
<td>111 (62.4%)</td>
<td>105 (48.4%)</td>
<td>.006</td>
<td>2.20 (1.19-3.37)</td>
</tr>
<tr>
<td>Previous vascular lesions, yes (any)</td>
<td>136 (76.4%)</td>
<td>154 (71.0%)</td>
<td>.252</td>
<td>–</td>
</tr>
<tr>
<td>Stroke diagnose</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic stroke</td>
<td>161 (90.4%)</td>
<td>205 (94.5%)</td>
<td>.127</td>
<td>–</td>
</tr>
<tr>
<td>Intraparenchymal hemorrhage</td>
<td>15 (8.4%)</td>
<td>10 (4.6%)</td>
<td>.121</td>
<td>–</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>1 (0.6%)</td>
<td>1 (0.5%)</td>
<td>.888</td>
<td>–</td>
</tr>
<tr>
<td>Cerebral venous thrombosis</td>
<td>1 (0.6%)</td>
<td>1 (0.5%)</td>
<td>.888</td>
<td>–</td>
</tr>
<tr>
<td>Oxford Stroke Classification (TACI vs others)</td>
<td>38 (23.6%)</td>
<td>4 (2%)</td>
<td>&lt;.001</td>
<td>15.52 (5.41-44.56)</td>
</tr>
<tr>
<td><strong>Topography</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supratentorial (vs infratentorial)</td>
<td>121 (83.4%)</td>
<td>142 (87.1%)</td>
<td>.363</td>
<td>–</td>
</tr>
<tr>
<td>Verteobasilar territory (vs carotid territory)</td>
<td>18 (12.4%)</td>
<td>11 (6.7%)</td>
<td>.089</td>
<td>–</td>
</tr>
<tr>
<td>MCA (vs other)</td>
<td>96 (65.8%)</td>
<td>95 (58.3%)</td>
<td>.177</td>
<td>–</td>
</tr>
<tr>
<td>Right hemisphere (vs left)</td>
<td>60 (33.7%)</td>
<td>77 (35.5%)</td>
<td>1.000</td>
<td>–</td>
</tr>
<tr>
<td>Stroke lesion volume (cc)</td>
<td>46.8 ± 112.6</td>
<td>8.8 ± 21.1</td>
<td>&lt;.001</td>
<td>1.02 (1.01-1.03)</td>
</tr>
</tbody>
</table>
TABLE 3 Multivariate analysis for oropharyngeal dysphagia (OD) risk factors. OR and 95% CI are shown for those variables that were significantly associated with OD (P < .05)

<table>
<thead>
<tr>
<th>Sociodemographic characteristics</th>
<th>P-value</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>&lt;.001</td>
<td>1.05 (1.02-1.08)</td>
</tr>
<tr>
<td>Sex</td>
<td>0.163</td>
<td>0.65 (0.35-1.92)</td>
</tr>
<tr>
<td>Widowed</td>
<td>0.991</td>
<td>1.00 (0.52-1.95)</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Clinical status before admission</th>
<th>P-value</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>0.422</td>
<td>1.33 (0.66-2.69)</td>
</tr>
<tr>
<td>Previous cerebral infarction</td>
<td>0.050</td>
<td>2.40 (1.00-5.79)</td>
</tr>
<tr>
<td>Major cardioembolic heart disease</td>
<td>0.357</td>
<td>0.71 (0.35-1.47)</td>
</tr>
<tr>
<td>mRS &gt;1</td>
<td>0.891</td>
<td>1.05 (0.51-2.15)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical data on admission</th>
<th>P-value</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NIHSS &gt;6</td>
<td>0.002</td>
<td>3.52 (1.57-7.87)</td>
</tr>
<tr>
<td>TACI</td>
<td>0.093</td>
<td>2.88 (0.84-9.94)</td>
</tr>
<tr>
<td>Stroke lesion volume</td>
<td>0.004</td>
<td>1.02 (1.01-1.03)</td>
</tr>
</tbody>
</table>

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

patients still had OD, which implies that one-third of patients admitted to our hospital with stroke were discharged with OD. The natural history and evolution of poststroke OD is still unknown and there is a debate on the spontaneous improvement of OD. These results clearly show that the prevalence of poststroke OD is still very high on hospital discharge.

The test we used to determine prevalence of dysphagia after stroke is another strength of the study. We used the V-VST, a validated clinical method with high sensitivity and specificity values (0.94 and 0.88, respectively) for detecting OD, enabling us to estimate the true prevalence of dysphagia after stroke. However, we should consider that the V-VST is not a gold standard instrumental assessment and thus the prevalence observed is not the exact one. Our estimation of the true prevalence of OD according to the sensitivity and specificity of the V-VST in this study is 40.3%. We used the V-VST because it was technically and ethically inappropriate to perform a VFS or FEES on all acute stroke patients.

In summary, our results show high prevalence of poststroke OD in patients with mild and moderate stroke from a medium-sized general hospital; this is a highly reproducible model across Europe.

4.2 Risk factors for presenting OD after stroke

The second important result that we present is that stroke severity and patient status before the stroke episode are better determinants for presenting OD after stroke than lesion location. We found that older age, previous stroke diagnosis, high NIHSS score, and large stroke lesion volume were independent risk factors for OD. Previous studies have analyzed factors related to OD in stroke, but most of them with few variables in the statistical analysis and, in some cases, only univariate analysis. Previous studies including multivariate analysis present heterogeneous results. Age and severity of stroke have been reported as independent factors after multivariate analysis. Paciaroni et al. reported that advanced age and NIHSS >15 was associated with OD. Flowers et al. also found a close relationship between OD and severity of stroke using Canadian Severity Scale. Previous stroke diagnosis has been reported as a risk factor for poststroke OD in several studies but our study is the first one that reports it as an independent risk factor.

In contrast, no neurotopographic variable was significantly associated with OD. This can be explained by the multifocal and bilateral control of swallowing in the brain; the functions lost from small localized lesions can be assumed and compensated by unaffected areas of the brain. On the other hand, large or multiple lesions (from the current or a previous infarct) in an “old” brain are more likely to cause dysphagia. Another important result from our study was that the level of consciousness, aphasia, dysarthria and visual field affection were independent risk factors for OD.

Stroke may affect afferent or efferent neuronal circuits participating in deglutition. The integrity of afferent pathways from the oropharynx to the cortex can be assessed using electroencephalography through sensory-evoked potentials elicited by pharyngeal electrical stimulation. Efferent pathways from the cortex to the pharynx can be characterized using electromyography through motor-evoked potentials elicited by transcranial magnetic stimulation. Dysfunction in both cortico-mediated evoked responses is associated with delayed swallowing response and aspiration. Previous studies have reported hemispherical asymmetry in motor control of swallowing and the relevance of impaired oropharyngeal sensitivity on aspiration. In a previous independent study, we found that chronic poststroke OD was associated with stroke severity and the degree of leukoaraiosis present. In that study, we found that impaired conduction and cortical integration of pharyngeal sensory inputs at the stroke site were key features of poststroke OD. We also found that impaired safety of swallow in chronic poststroke patients was caused by specific impairments in the motor swallow response, including delayed timing of airway protection mechanisms and weak tongue propulsion forces.

4.3 OD as a prognostic factor

The third result we wish to highlight is that we found a close association between presenting OD after stroke and poor clinical prognosis. In our study, poststroke OD is an independent risk factor for prolonged length of hospital stay (up to 2 days more), and for institutionalization after hospital discharge and poorer functional status 3 months after discharge. These results agree with the study by Smithard et al. These three factors are of a great importance not only from the perspective of the patient health, but also because they represent a major social and economic burden. A study on the cost of stroke in Spain found that acute-care hospitalization costs closely correlated with length of stay (R = .847, P < .001), so having OD increases the healthcare costs of stroke. It has also been reported that the economic burden of stroke care (healthcare and non-healthcare costs) is directly related to the level
TABLE 4 Multivariate analysis for stroke clinical complications. Data is presented as n and % of available data, except for length of stay (mean ± SD). Adjusted OR (or HR in case of mortality) include all the sociodemographic and clinical data that showed a P < .05 in the univariate analysis (see complete multivariate models in supplementary material)

<table>
<thead>
<tr>
<th>Clinical data on discharge</th>
<th>OD patients (n = 178)</th>
<th>Non-OD patients (n = 217)</th>
<th>Univariate P-value</th>
<th>OR (95% CI)</th>
<th>Adjusted P-value</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurological complications</td>
<td>25 (14.0%)</td>
<td>10 (4.6%)</td>
<td>&lt;.001</td>
<td>3.38 (1.58-7.25)</td>
<td>.519</td>
<td>1.44 (0.47-4.37)</td>
</tr>
<tr>
<td>Respiratory infections</td>
<td>21 (11.8%)</td>
<td>3 (1.4%)</td>
<td>&lt;.001</td>
<td>9.54 (2.80-32.55)</td>
<td>.873</td>
<td>1.14 (0.22-5.87)</td>
</tr>
<tr>
<td>Urinary infections</td>
<td>12 (6.7%)</td>
<td>2 (0.9%)</td>
<td>.002</td>
<td>7.77 (1.72-35.2)</td>
<td>.461</td>
<td>2.61 (0.20-33.52)</td>
</tr>
<tr>
<td>Length of stay (days)</td>
<td>8.2 ± 5.0</td>
<td>6.1 ± 2.9</td>
<td>&lt;.001</td>
<td>2.11*</td>
<td>.049</td>
<td>0.938*</td>
</tr>
<tr>
<td>Mortality</td>
<td>20 (11.2%)</td>
<td>1 (0.5%)</td>
<td>&lt;.001</td>
<td>27.34 (3.63-205.87)</td>
<td>†</td>
<td>†</td>
</tr>
<tr>
<td>Destination on discharge</td>
<td>76 (49.0%)</td>
<td>176 (83.8%)</td>
<td>&lt;.001</td>
<td>0.186 (0.12-0.30)</td>
<td>.027</td>
<td>0.47 (0.24-0.92)</td>
</tr>
</tbody>
</table>

Clinical data at 3 months follow-up

| mRS >1           | 138 (77.5%) | 71 (33.5%) | <.001 | 6.85 (4.4-10.8) | <.001 | 3.00 (1.58-5.68) |
| Readmissions     | 34 (21.7%)  | 57 (26.6%) | .329  | 0.83 (0.51-1.35) | —     | —               |
| Respiratory infections | 12 (6.7%) | 7 (3.2%)   | <.001 | 4.87 (2.25-10.54) | .784  | 1.15 (0.42-3.17) |
| Mortality        | 41 (28.7%)  | 3 (1.9%)   | <.001 | 17.46 (5.39-56.51) | .011  | 6.90 (1.57-30.34) |

Clinical data at 12 months follow-up

| Readmissions     | 75 (47.8%)  | 117 (54.7%) | .208  | 0.78 (0.50-1.34) | —     | —               |
| Respiratory infections | 45 (25.6%) | 28 (13.1%) | .003  | 2.28 (1.35-3.85) | .393  | 1.37 (0.665-2.818) |
| Mortality        | 55 (31.1%)  | 7 (3.2%)    | <.001 | 11.40 (5.19-25.04) | .202  | 1.97 (0.696-5.56) |

All models were adjusted by mRS before admission, and stroke lesion volume. Additionally, each model was adjusted for the following variables: (a) age, NIHSS on admission, TACI and stroke lesion volume; (b) age, marital status, heart disease, NIHSS on admission, TACI and stroke lesion volume; (c) age, sex, marital status, abode before admission, major cardioembolic heart disease, NIHSS on admission, TACI and stroke lesion volume; (d) age, marital status, abode before admission, heart disease, NIHSS on admission, TACI, stroke lesion volume and neurologic complications; (e) age, sex, abode before admission, major cardioembolic heart disease, NIHSS on admission, TACI, stroke lesion volume, respiratory infection and neurologic complications; (f) age, sex, marital status, abode before admission, major cardioembolic heart disease, previous infarct, NIHSS on admission, TACI, stroke lesion volume and neurologic complications; (g) age, marital status, heart disease, NIHSS on admission, TACI and stroke lesion volume; (h) marital status, mRS before admission, major cardioembolic heart disease, respiratory infection and stroke lesion volume; (i) age, marital status, NIHSS on admission and major cardioembolic heart disease.

*Beta coefficient.
†It was not possible to perform the analysis as there was only one case of mortality among patients without OD.

of associated disability, so poorer patient functional status, such as in dysphagic patients, also means higher total costs. OD is also a commonly reported risk factor for the development of pneumonia and respiratory infections following stroke, in both the acute and rehabilitation settings. In the univariate analysis, we found that OD was associated with a 5-fold increase in respiratory infections during hospital stay, 5-fold increase in the first 3 months after stroke, and twice as many at 1-year follow-up. As reported by Smithard et al., the association did not remain significant when adjusted for other confounding variables. A possible explanation for this is that the development of aspiration pneumonia is multifactorial. Although the presence of aspiration and impaired swallowing safety is a sine qua non-condition for the development of the infection, it also depends on several factors such as the nutritional and functional status of the patient, immunological alterations, age, oral health status and oral colonization by respiratory pathogens. Moreover, a systematic screening and management program of OD, such as that implemented in our hospital, can reduce the prevalence of pulmonary infections in OD patients, as reported in other studies.

4.4 | OD is an independent mortality risk factor

Finally, we found that OD after stroke was a strong risk factor of mortality, as found in previous studies. During acute care, the likelihood of a patient with OD dying is 27 times higher than in a patient that does not have OD. Although we could not perform a multivariate adjustment on this data (because only one patient without dysphagia died), the clinical relevance of this finding is enormous. In addition, we found that OD was an independent risk factor of
mortality 3 months after stroke. At 1-year follow-up, OD remained a significant risk factor in the univariate analysis, but the significance disappeared when adjusting for the confounding variables. This point highlights the relevance for early intervention of stroke-associated OD as most of the deaths attributable to dysphagia occur during hospital stay and in the first 90 days after the stroke episode.

Our study has several limitations. As previously discussed, our hospital is a Primary Stroke Center, which means that severe stroke patients are transferred to a Comprehensive Stroke Center where they are admitted during the acute phase and then discharged home or transferred to our hospital. This was the reason 73 patients were excluded, most of them with severe stroke. If we had included them, the median NIHSS and the prevalence of OD would have probably been higher.

Second, this is an observational study based upon daily clinical practice. For example, MRI was only performed when the neurologist considered it necessary. However, in our opinion, this is also one of the strengths of the study; it was prospective and included all acute strokes admitted to our hospital from the emergency department, without selection bias. Another important issue related to the daily clinical practice in our hospital is the fact that the detection and management of OD patients itself could modify the outcome of these patients; eg, the fluid viscosity adaptation itself can significantly reduce the risk of respiratory infections in OD patients.20

In conclusion, the prevalence of new-onset dysphagia was 45.06% after a stroke. Stroke severity and patient status before the stroke episode are better determinants of OD than lesion location. Presenting OD after stroke is associated with high mortality rates and poor clinical prognosis (prolonged length of hospital stay, institutionalization after hospital discharge and poor functional status 3 months after discharge). Our results suggest that systematic screening programs and early OD management could significantly improve poststroke patient outcome. Treatment is also changing from compensatory strategies to promoting brain plasticity, both to recover swallow function, and to improve brain-related swallowing dysfunction.26

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DISCLOSURE

Laia Rofes, Desiree Muriana, Ernest Palomeras, Natalia Vilardell1, Elisabet Palomera, Daniel Alvarez-Berdugo, and Virginia Casado report no disclosures. Pere Clavé president of the European Society of Swallowing Disorders.

AUTHOR CONTRIBUTION

LR designed the study, contributed to data interpretation, did the literature search, created the figures and wrote the first draft of the paper; DM designed the study, did the literature search, recruited patients, collected clinical data, contributed to data interpretation; EP designed the study, recruited patients, collected clinical data, contributed to data interpretation; NV collected clinical data, contributed to data interpretation; EP performed the statistical analysis; DA-B collected clinical data; VC recruited patients, collected clinical data; PC designed the study, contributed to data interpretation. All the authors: critically revised and approved the final version of the paper.

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