Predictive Factors of Swallowing Disorders and Bronchopneumonia in Acute Ischemic Stroke

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Background: In stroke patients, early complications such as swallowing disorders (SD) and bronchopneumonia (BP) are frequent and may worsen outcome. The aim of this study was to evaluate the prevalence of SD in acute ischemic stroke (AIS) and the risk of BP, as well as to identify factors associated with these conditions. *Methods*: We retrospectively studied all AISs over a 12-month period in a single-center registry. We determined the frequency of SD in the first 7 days and of BP over the entire hospital stay. Associations of SD and BP with patient characteristics, stroke features, dental status, and presence of a feeding tube were analyzed in multivariate analyses. *Results*: In the 340 consecutive patients, the overall frequency of SD and BP was 23.8% and 11.5%, respectively. The multivariate analyses showed significant associations of SD with NIHSS scores >4, involvement of the medulla oblongata and wearing a dental prosthesis (area under the receiver-operator curve (AUC) of 76%). BP was significantly associated with NIHSS scores >4, male sex, bilateral cerebral lesions, the presence of SD, and the use of an enteral feeding tube (AUC 84%). In unadjusted analysis, unfavorable 12-month outcome and mortality were increased in the presence of SD. *Conclusion*: In AIS, SD and BP are associated with stroke severity and localization and wearing a dental prosthesis increases the risk of SD. Given that patients with SD have an increased risk of poor outcome and mortality, high-risk patients warrant early interventions, including more randomized trials.

Key Words: Swallowing disorders—dysphagia—acute ischemic stroke—bronchopneumonia—speech therapy—predictive factors

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Introduction

Swallowing disorders (SD) and bronchopneumonia (BP) are frequent problems in the acute phase of stroke, prolonging hospital stay and may alter a patient's long-term outcome and mortality.[1,2] The frequency of these stroke complications is linked to several factors such as the type of stroke, the location of the cerebral lesion, the age or the associated comorbidities.[1–4]

Although there is no international consensus on the choice of SD screening,[1,5] the early identification and multidisciplinary management of SD during the first weeks after stroke are key components in acute stroke unit care [6] and for improved outcome.[5,7,8] SD increase the risk of BP related to aspiration,[1,2] but are not the only factors responsible. Comorbidities[9] and alteration of the immune system in the acute stroke phase[10] also play important roles. Nevertheless, in order to reduce the risk of respiratory infections, the management of SD includes rehabilitation with a speech therapist.

Several studies identified factors associated with the risk of developing SD or BP after stroke, but few have applied methods using multivariate analysis.

The aim of this study was to assess the prevalence of SD in consecutive acute ischemic stroke (AIS) patients in the first 7 days of hospitalization and estimate the risk of developing BP over the entire hospital stay. Furthermore, we evaluated the mortality rate in cases of SD and BP and identified predictors of SD and BP.

Materials and Methods

We carried out a retrospective study of 340 AIS patients admitted consecutively to the Stroke Unit and/or intensive care service of the Lausanne University Hospital (CHUV), between January 1 and December 31, 2015. We extracted data from the ASTRAL registry (Acute STroke Registry and Analysis of Lausanne[11]) that collects, in a prespecified manner, all patients admitted within 24 hours of their last known well time, including recurrent ischemic strokes. In ASTRAL, a large amount of demographic data (age, sex), risk factors (hypertension, smoking, diabetes, hyperlipidemia, body mass index [BMI]), past cerebrovascular events, and acute clinical data (admission NIHSS score, clinical deficits) are recorded. Also, vascular territories and cerebral structures affected, as well as stroke mechanism according to the TOAST classification[12] are registered. In all AIS patients retrieved from ASTRAL, the medical records and notes of the swallowing therapist and the nurse were reviewed to identify the presence of a dental prosthesis, SD, BP, nasogastric tube feeding (NGT) and percutaneous gastrostomy (PEG) insertion during the hospitalization (Table 1).

All AIS patients in our institution undergo a standardized bedside SD screening test from the patient nurse within 24 hours of admission, unless bronchopneumonia was apparent. The test is DePippo's[13] modified version of the Burke Dysphagia Screening Test by and consists of swallowing 90 mL of water and watching for coughs or voice change after swallowing. If patients cough or have a hoarse voice, we consider the test as failed. If unsuccessful, SD is considered present and the patient is kept nil-by-mouth, with the test repeated the next day. If patients do not cough or change voice at test 1, we repeat the test. If the second test shows no changes, we consider the patient without SD, but repeat the test in cases of clinical worsening. For oral feeding of patients who do not pass a second test, thickened liquids are tried; if successful swallowing occurs, these patients are given food with adapted texture.

For this study, we considered patients had SD if test 1 or 2 of the swallowing screening test showed cough or voice change (pathological), if the patients spontaneous complain of a swallowing disorders without swallowing screening test and if their food texture had to be adapted, or if they required specialized speech therapy in their first 7 days of hospitalization; our standardized protocol involved a speech therapy upon the dysphagia screening is pathological.

We identified BP retrospectively in medical records. We assigned BP if the diagnosis was present in medical charts or discharge letters; in our stroke center, we diagnose BP as presence of "new lung infiltrate plus clinical evidence that the infiltrate is of an infectious origin, which includes the new onset of fever, purulent sputum, leukocytosis, and decline in oxygenation."[14]

Rankin-certified personnel assessed Rankin score, including mortality at 12 months in a structured telephone interview, using the modified Rankin score (mRs). We considered outcome was favorable if the difference between long-term (12 months) and prestroke mRS was $\leq$2 for the corrected mRS.

A statistician (M.F.) performed the statistical analyses using STATA 14.1 (StataCorp. 2017. Stata Statistical Software: Release 15. College Station, TX: StataCorp LLC). We describe correlation of SD and BP with demographic and clinical variables, risk factors, and localization with the odds ratio (OR), its confidence intervals at 95% (95% CI) and the P value. We performed logistic regression with all variables having $P < .20$ in the univariate comparison. We tested retained multivariable models with the "Pearson or Hosmer-Lemeshow goodness-of-fit test." Finally, we evaluated the power of prediction for the complications of each model using the area under the receiver-operator curve (AUC) (Figs 1 and 2). We set a P value $<.05$ as significant in the multivariate analyses.

The local ethics committee (Canton Vaud, Switzerland) approved the study protocol on 30.05.16 (project ID: 2016-00523). Since it is often not possible to obtain informed consent in patients with acute cerebrovascular diseases due to acute or pre-existing cognitive impairment (cognitive/dementia), the principle of "lack of consent" according to Art. 34 HRL applies (Human Research Law, Lack of Consent

Table 1. *Baseline characteristics and 12-month outcomes of study patients*

Total, N = 340	Without SD	With SD	With BP	SD and BP
Number of patients, n (%)	259 (76.2)	81 (23.8)	39 (11.5)	24 (7.1)
Age years (Median ± IQR)	75.3 ± 18.9	77.8 ± 18.7	80.7 ±16.2	77.2 ± 20.0
Sex (women)	115 (44.4)	41 (50.6)	12 (30.8)	7 (29.2)
Hypertension	192 (74.1)	65 (80.3)	35 (89.7)	22 (91.2)
Diabetes	52 (20.1)	16 (19.8)	9 (23.1)	3 (12.5)
Hyperlipidemia	202 (78.0)	64 (79.0)	32 (82.1)	19 (79.2)
Current smoking	47 (18.4)	19 (23.8)	10 (27.0)	9 (37.5)
Atrial fibrillation	74 (28.6)	41 (50.6)	21 (53.9)	15 (62.5)
Previous clinical stroke (or TIA), n (%)	58 (23.6)	14 (17.8)	8 (21.1)	6 (26.1)
BMI (kg/m^2) (Median ± IQR)	25.4 ± 6.9	25.0 ± 7.0	25.9 ± 8.0	24.5 ± 6.0
NIHSS at admission, n (%)				
(Median ± IQR)	5.0 ± 10.0	11.5 ± 12.0	14.0 ± 12.0	13.0 ± 13.5
[0-4]	113 (45.0)	10 (12.8)	1 (2.6)	1 (4.2)
[5-15]	95 (37.9)	39 (50.0)	22 (56.4)	12 (50.0)
>15 [16-36]	43 (17.1)	29 (37.2)	16 (41.0)	11 (45.8)
Mechanism (modified TOAST), n (%)				
Atherosclerosis	32 (14.5)	12 (16.0)	6 (17.1)	4 (18.2)
Cardioembolism	75 (33.9)	32 (42.7)	16 (45.7)	11 (50.0)
Small-vessel occlusion	19 (8.6)	4 (5.3)	1 (2.9)	1 (4.6)
Other determined etiology	24 (10.9)	9 (12.0)	3 (8.6)	2 (9.1)
Undetermined etiology	71 (32.1)	18 (24.0)	9 (25.7)	4 (18.2)
Side of stroke, n (%)				
Bilateral	24 (9.6)	7 (8.8)	8 (21.6)	6 (26.1)
Vascular territory, n (%)				
Anterior circulation	170 (71.4)	63 (80.8)	28 (73.7)	16 (69.6)
Posterior circulation	51 (21.4)	10 (12.8)	7 (18.4)	4 (17.4)
Simultaneous anterior and posterior	9 (3.8)	4 (5.1)	3 (7.9)	3 (13.0)
Cerebral structure affected, n (%)				
Frontal lobe	121 (60.2)	47 (72.3)	19 (67.9)	13 (68.4)
Temporal lobe	77 (38.1)	37 (56.9)	16 (57.1)	12 (63.2)
Parietal lobe	110 (54.5)	46 (70.8)	20 (71.4)	15 (79.0)
Occipital lobe	24 (11.7)	5 (7.8)	5 (17.2)	3 (16.7)
Thalamic	18 (8.8)	3 (4.7)	1 (3.7)	1 (5.6)
Mesencephalic	15 (7.4)	5 (7.7)	4 (14.3)	4 (21.1)
Pontic	13 (6.4)	6 (9.2)	5 (17.9)	4 (21.0)
Medulla oblongata	2 (1)	4 (6.3)	3 (11.1)	2 (11.1)
Cerebellar	18 (8.9)	8 (12.3)	4 (14.3)	3 (15.8)
Multiple territories	9 (4.5)	5 (7.7)	3 (10.7)	3 (15.8)
Undetermined localization (mostly lacunar strokes)	10 (5.0)	1 (1.6)	0 (0)	0 (0)
Dental prosthesis, n (%)	10 (3.9)	13 (16.1)	5 (12.8)	4 (16.7)
Swallowing disorders, n (%)	-	81 (100)	24 (61.5)	24 (100)
NGT or PEG, n (%)	11 (4.3)	45 (55.6)	20 (51.3)	18 (75.0)
In hospital mortality, n (%)	22 (8.5)	7 (8.9)	6 (15.8)	4 (17.4)
Unfavorable outcome at 12 months	78 (33.8)	54 (74.0)	29 (82.9)	19 (86.4)
Mortality at 12 months	49 (21.2)	26 (35.6)	18 (51.4)	11 (50.0)

Abbreviations: BMI, body mass index; BP, bronchopneumonia; IQR, interquartile range; NIHSS, National Institutes of Health Stroke Scale; SD, swallowing disorders; TOAST, Trial of Org 10172 in Acute Stroke Treatment.

Percent refers to recorded values only and missing values were omitted.

or Information, Federal Law - Switzerland); any refusal by the data subject has been respected. In 2007, the ethic committee accepted the use of clinical, radiological, and biological data from patients with AIS collected for routine management in the Cerebrovascular Centre of the ASTRAL Registry for research purposes without written consent.

Results

Over the 12-month observation period, we extracted 340 patients with AIS from ASTRAL for this analysis. One hundred and fifty-seven were women (45.9%), and the median age was 75 years (IQR = 18 with a range of 21-96

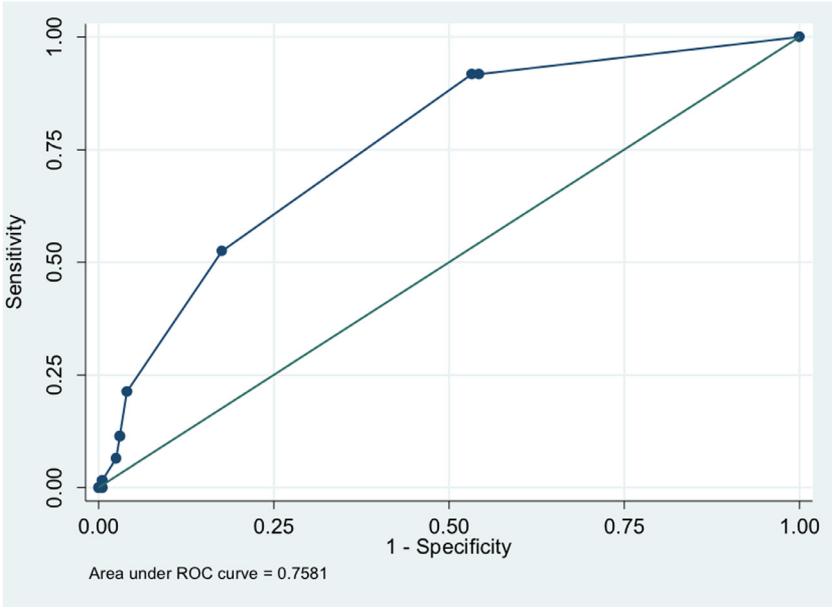

Figure 1. *ROC curve for factors associated with swallowing disorders (SD).*

years). Median length of hospital stay was 8 days (IQR = 9), and ranged from 0-50 days.

One hundred and forty-eight patients (43.2%) had a standardized swallowing test documented in the medical record, of which 51 (51/148 = 34.5%) were pathological. Adding other swallowing difficulty criteria (adaptation of food texture, logopedic treatment), 81 patients (23.6%) suffered SD. Seventy-four of these SD patients (91.4%) received swallowing therapy from a logopedist during their hospitalization. We list other baseline variables all patients in Table 1.

Of the 39 patients (11.5%) with documented BP over the entire hospital stay, there are 24/39 (61.5%) with SD and of which 22/24 patients (91.7%) patients with SD and BP had a logopedic treatment, while the 17 (17/39) other BP patients had no treatment.

On the other side, they were 81 patients with SD whose 74 received a logopedic treatment for SD. Among the 7 SD patients who did not receive treatment (81 SD minus 74 logopedist treated), 2 out of them had BP and 2 were in palliative care. The remaining 3 of 7 SD patients without logopedic treatment were transferred to other hospitals

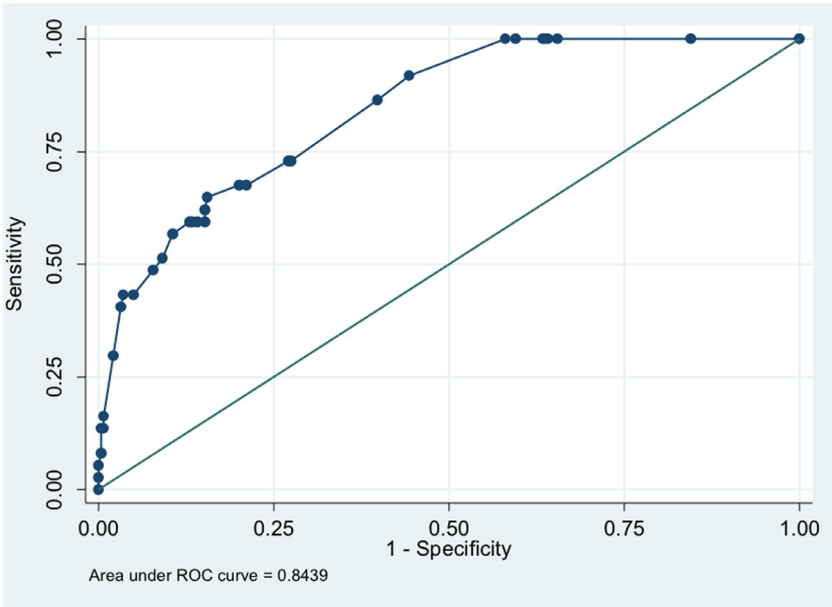

Figure 2. *ROC curve for factors associated with bronchopneumonia (BP).*

before we initiated treatment. Patients with BP and SD had a higher mean NIHSS score in univariate analysis, compared to those without SD (14.6 ± 8.5 versus 13.2 ± 6.2) and their main stroke-cause was cardioembolic.

A NGT was inserted at least once in 56 patients (16.5%) and a PEG was used before discharge in 2 (2/340 = .6%) patients.

Twenty-nine patients (8.6%) died from this cohort during hospitalization, 7 of the patients had SD and 6 BP.

According to our multivariate analysis (Table 2), the variables associated with increased risk of SD are a moderate-to-severe neurological deficit on admission (NIHSS score between 5 and 15 (OR 6.17, $P < .001$) or above 16 (OR 9.35; $P < .001$), wearing a dental prosthesis (OR 3.04; $P < .038$), involvement of the medulla oblongata (OR 20.11; $P = .003$). The model had an AUC of 75.8%.

Table 3 shows the multivariate analysis for factors associated with BP. We found significant association for male sex (OR 2.33, $P = .043$), moderate-to-severe neurological deficit on admission (OR 21.16, $P = .004$; above 16, OR 23.14, $P = .004$), bilateral cerebral lesions (OR 4.13, $P = .013$), SD (OR 3.40, $P = .010$), and presence of a NGT or PEG (OR 2.4). In this multivariate model, the AUC was 84.4%.

Unadjusted 12-month functional outcome was more often unfavorable in patients with SD (OR 5.6, $P = .014$) and mortality was higher (OR 2.1, $P < .001$), and even more in patients with BP (unfavorable outcome: OR 7.8, $P < .001$; mortality OR 3.9, $P < .001$).

Discussion

In a retrospective analysis using a standardized SD protocol, we found a SD frequency of 23.6% in the first 7 days after admission and a BP frequency of 11.5%.

In multivariate analyses, SD and BP were associated with several factors, such as stroke severity and localization. SD was also associated with having a dental prosthesis. Twelve-month outcome was clearly better without SD in terms of function and mortality, confirming previous studies. In addition, our findings support that an early screening for SD would prevent BP after AIS, and thus reduce the proportion of mortality associated with BP and SD in poststroke recovery, knowing that other causes can cause death in these multimorbidities patients.[15,2]

The prevalence of SD and BP is consistent with previous studies based on clinical screening.[1,16,17] Our results are lower than studies which used technical screening methods that are more sensitive for SD.[4] These data emphasize the necessity to assess dysphagia in the early phase of acute stroke in order to put into place measures to reduce its impact on the patient.[15,18–20]

Previous studies have described the statistical association between SD and BP and stroke severity,[15,19,21–23] and our multivariate analyses lend further support to this association.

The previous authors' views on the correlation of the topography of stroke lesions and SD or BP are controversial.[16,24,25,19,22] Our multivariate analyses specifically highlight a statistical association of medullary lesions with SD.[16,24] This seems plausible, given the importance of this structure in the execution and control of swallowing.

In our study, and in contrast to previous studies,[26,13,27] there is no significant association between the dysphagia and the side of the brain affected by the stroke. However, we found a significant correlation between BP and bilateral cerebral lesions, independent of stroke severity. The likely explanation for this observation is that most of the central mechanisms involved in swallowing have bilateral input allowing for better compensation of dysphagia in unilateral lesions, but inversely leads to worse dysphagia if compensation from the other side of the brain is deficient.

Although other studies have described a significantly higher risk of BP in male patients,[19] its reasons remain

Table 2. *Factors associated with swallowing disorders in the multivariate analysis*

	Without SD versus With SD		
Variable	OR	95% CI	*P Value*
NIHSS at admission			
0-4 (reference)	-	-	-
5-15	6.2	2.4-15.6	<.001***
>15	9.3	3.4-25.4	<.001***
Medulla oblongata involvement	20.1	2.9-140.9	.003**
Dental prosthesis	3.0	1.1-8.7	.038*
AUC for SD prediction: .76			

Abbreviations: AUC, area under cover; 95% CI, 95% confidence interval; NIHSS, National Institutes of Health Stroke Scale; OR, odds ratio; SD, swallowing disorders.

Asterisks denote statistical significance.

*$P < .05$.

**$P < .01$.

***$P < .001$.

Table 3. *Factors associated with bronchopneumonia in the multivariate analysis*

	Without BP versus with BP		
Variable	OR	95% CI	*P Value*
Male sex	2.3	1.0-5.3	.043*
NIHSS at admission			
[0-4]	-	-	-
[5-15]	21.2	2.6-170.7	.004**
>15 [16-36]	23.1	2.8-193.6	.004**
Bilateral cerebral lesion	4.1	1.4-12.6	.013*
Swallowing disorders	3.4	1.3-8.6	.010*
NGT or PEG	2.4	.9-6.4	.074
AUC for BP prediction: .84			

Abbreviations: AUC, area under cover; BP, bronchopneumonia; 95%CI, 95% confidence interval; NGT, nasogastric tube; NIHSS, National Institutes of Health Stroke Scale; OR, odds ratio; PEG, percutaneous gastrostomy.

Asterisks denote statistical significance.

*$P < .05$.

**$P < .01$.

speculative. Discussed hypotheses are protective effects of female hormones on the immune response and the differential distribution of risk factors between sexes.[21]

In univariate but not multivariate analysis, we found that use of enteral feeding tubes was associated with BP, as previously described.[1,2,28] Routine early enteral feeding is not recommended according to the FOOD trials 2 and 3, and our policy is to wait at least 5-7 days before NGT placement for feeding, unless essential drugs need enteral administration. For late enteral feeding in persistent dysphagia, placement of PEG seems superior to NGT, if a patient needs support for more than 3 weeks.[29,30]

In our study, we showed that patients with dental prostheses are at higher risk for SD in multivariate analyses. The reason for this observation could be that having a dental prosthesis is an indicator of higher fragility and SD risk; alternatively, swallowing with a prosthesis in subacute stroke may be more difficult than with one's own teeth, or, patients did not wear it while hospitalized leading to altered mastication and swallowing process. To our knowledge, other studies have not reported an impact of dental prostheses on SD in the acute phase after stroke.

Our study has several limitations, including being a retrospective study using a stroke registry and additional information from medical records that may lead to underreporting of SD and BP. Second, documentation of a standardized swallowing assessment was missing in more than half of our patients, again leading to potential underestimation of SD. The main reason for this lack of a formalized assessment was absence of suspicion of SD by the nurses or physicians, and the intervention of speech therapists without screening in case of an SD complaint from the patient. It has been shown, however, that standardized swallowing assessment is superior to simple clinical observation,[15] and leads to better outcomes.[6] Finally, we did not evaluate the long-term course of SD in our patients.

Conclusion

In conclusion, our study identified 3 factors associated with risk of SD and 5 with BP. All these factors are nonmodifiable except for tube feeding and; whether the latter is a cause or consequence of SD and BP remains open.

Nonetheless, knowledge of these factors may allow clinicians to identify readily AIS patients at high risk of developing these complications to intensify their monitoring and early interventions to prevent them. Still, the number of randomized trials to identify effective early treatment is very limited and we now need such trials to guide best clinical practice.

Conflicts of Interest

All authors state they have no conflict of interest to disclose.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jstrokecerebrovasdis.2019.04.025.

References

1. Arnold M, Liesirova K, Broeg-Morvay A, et al. Dysphagia in acute stroke: incidence, burden and impact on clinical outcome. PLoS One 2016;11:e0148424.
2. Brogan E, Langdon C, Brookes K, et al. Dysphagia and factors associated with respiratory infections in the first week post stroke. Neuroepidemiology 2014;43:140-144.
3. Brogan E, Langdon C, Brookes K, et al. Respiratory infections in acute stroke: nasogastric tubes and immobility are stronger predictors than dysphagia. Dysphagia 2014;29:340-345.
4. Martino R, Foley N, Bhogal S, et al. Dysphagia after stroke: incidence, diagnosis, and pulmonary complications. Stroke 2005;36:2756-2763.

5. Daniels SK, Anderson JA, Willson PC. Valid items for screening dysphagia risk in patients with stroke: a systematic review. Stroke 2012;43:892-897.
6. Urimubenshi Gerard LP, Cadilhac Dominique A, Kagwiza Jeanne N, et al. Association between patient outcomes and key performance indicators of stroke care quality: a systematic review and meta-analysis. Eur Stroke J 2017;2:287-307.
7. González-Fernández M, Ottenstein L, Atanelov L, et al. Dysphagia after Stroke: an overview. Curr Phys Med Rehabil Rep 2013;1:187-196.
8. Ramsey DJ, Smithard DG, Kalra L. Early assessments of dysphagia and aspiration risk in acute stroke patients. Stroke 2003;34:1252-1257.
9. Langmore SE, Terpenning MS, Schork A. Predictors of aspiration pneumonia: how important is dysphagia? Dysphagia 1998;13:69-81.
10. Chamorro Á, Meisel A, Planas AM, et al. The immunology of acute stroke. Nat Rev Neurol 2012;8:401-410.
11. Michel P, Odier C, Rutgers M, et al. The Acute STroke Registry and Analysis of Lausanne (ASTRAL): design and baseline analysis of an ischemic stroke registry including acute multimodal imaging. Stroke 2010;41:2491-2498.
12. Adams HP, Bendixen BH, Kappelle LJ, 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;24:35-41.
13. DePippo KL, Holas MA, Reding MJ. The Burke dysphagia screening test: validation of its use in patients with stroke. Arch Phys Med Rehabil 1994;75:1284-1286.
14. Kalil AC, Metersky ML, Klompas M, et al. Management of adults with hospital-acquired and ventilator-associated pneumonia: 2016 Clinical Practice Guidelines by the Infectious Diseases Society of America and the American Thoracic Society. Clin Infect Dis 2016;63:e61-e111.
15. Bray BD, Smith CJ, Cloud GC, et al. The association between delays in screening for and assessing dysphagia after acute stroke, and the risk of stroke-associated pneumonia. J Neurol Neurosurg Psychiatry 2017;88:25-30.
16. Daniels SK, Pathak S, Mukhi SV, et al. The relationship between lesion localization and dysphagia in acute stroke. Dysphagia 2017;32:777-784.
17. Paciaroni M, Mazzotta G, Corea F, et al. Dysphagia following Stroke. Eur Neurol 2004;51:162-167.
18. Cohen DL, Roffe C, Beavan J, et al. Post-stroke dysphagia: a review and design considerations for future trials. Int J Stroke 2016;11:399-411.
19. Henke C, Foerch C, Lapa S. Early screening parameters for dysphagia in acute ischemic stroke. Cerebrovasc Dis 2017;44:285-290.
20. Ickenstein GW, Riecker A, Höhlig C, et al. Pneumonia and in-hospital mortality in the context of neurogenic oropharyngeal dysphagia (NOD) in stroke and a new NOD step-wise concept. J Neurol 2010;257:1492-1499.
21. Kwon HM, Jeong SW, Lee SH, et al. The pneumonia score: a simple grading scale for prediction of pneumonia after acute stroke. Am J Infect Control 2006;34:64-68.
22. Okubo PC, Fábio SR, Domenis DR, et al. Using the National Institute of Health Stroke Scale to predict dysphagia in acute ischemic stroke. Cerebrovasc Dis 2012;33:501-507.
23. Ribeiro PW, Cola PC, Gatto AR, et al. Relationship between dysphagia, National Institutes of Health Stroke Scale Score, and predictors of pneumonia after ischemic stroke. J Stroke Cerebrovasc Dis 2015;24:2088-2094.
24. Flowers HL, Skoretz SA, Streiner DL, et al. MRI-based neuroanatomical predictors of dysphagia after acute ischemic stroke: a systematic review and meta-analysis. Cerebrovasc Dis 2011;32:1-10.
25. Hamdy S, Rothwell JC, Aziz Q, et al. Organization and reorganization of human swallowing motor cortex: implications for recovery after stroke. Clin Sci (Lond) 2000;99:151-157.
26. Broadley S, Croser D, Cottrell J, et al. Predictors of prolonged dysphagia following acute stroke. J Clin Neurosci 2003;10:300-305.
27. Hilker R, Poetter C, Findeisen N, et al. Nosocomial pneumonia after acute stroke: implications for neurological intensive care medicine. Stroke 2003;34:975-981.
28. Gomes GF, Pisani JC, Macedo ED, et al. The nasogastric feeding tube as a risk factor for aspiration and aspiration pneumonia. Curr Opin Clin Nutr Metab Care 2003;6:327-333.
29. Dennis M, Lewis S, Cranswick G, et al. FOOD: a multicentre randomised trial evaluating feeding policies in patients admitted to hospital with a recent stroke. Health Technol Assess 2006: 1-120. 10:iii-iv, ix-x.
30. Gomes CA, Andriolo RB, Bennett C, et al. Percutaneous endoscopic gastrostomy versus nasogastric tube feeding for adults with swallowing disturbances. Cochrane Database Syst Rev 2015. CD008096.