



# Incidence and Risk Factors for Dysphagia Following Non-traumatic Subarachnoid Hemorrhage: A Retrospective Cohort Study

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## Abstract

The aim of the study is to investigate dysphagia incidence and establish factors which may reliably predict dysphagia risk in individuals presenting with non-traumatic subarachnoid hemorrhage (SAH). A retrospective chart audit of 250 patients (151 females and 99 males) consecutively admitted with non-traumatic SAH to a major, tertiary neurosurgery referral center in Australia was conducted. Demographics, medical, and surgical information, along with speech–language pathology (SLP) assessment data were collected. Differences between dysphagic and non-dysphagic groups were evaluated using *t* tests,  $\chi^2$ , and Fisher’s exact tests. Univariate and multivariate logistic regression analysis was performed to establish factors associated with dysphagia risk. A total of 31.6% of participants were identified with dysphagia during acute inpatient admission based on SLP, medical officer, and/or nursing staff reports. Individuals with dysphagia had significantly ( $p < 0.01$ ) higher World Federation of Neurological Surgeons (WFNS) grading scores, were more likely to have an aneurysmal cause, were more likely to have secondary complications such as vasospasm, hydrocephalus, or new ischemia, were older, and had longer intubation and intensive care unit (ICU) periods than those without dysphagia. Dysphagia risk was significantly associated ( $p < 0.01$ ) with age  $> 57.5$  years, ICU length of stay  $> 7.5$  days, length of intubation  $> 1.5$  days, need for tracheostomy, vasospasm, and new stroke. Dysphagia is highly prevalent following non-traumatic SAH, and significantly associated with a number of factors. Established risk factors will improve current knowledge, promote early identification of dysphagia, and inform SLP referral criteria and management of this patient cohort.

**Keywords** Dysphagia · Subarachnoid hemorrhage · Incidence · Risk factors

## Introduction

Stroke is the third leading cause of mortality and contributes to significant disease morbidity in Australia [1]. Subsequently, \$5 billion is invested annually for the prevention and treatment of stroke, and national guidelines are

considered essential for the safe and effective provision of evidence-based stroke care [2, 3]. Non-traumatic subarachnoid hemorrhage (SAH) accounts for approximately 5% of all strokes and 500 deaths in Australia annually [1, 4]. Worldwide incidence of SAH is approximately 9.1 per 100,000 person-years [4]. Advances in SAH management have contributed to improved survival rates; however, high morbidity continues to impact physical, cognitive, and emotional domains of functioning [5]. It impacts individuals at an earlier age than other stroke subtypes, leading to greater loss of productive years [5–7]. There is a different presentation of neurological deficits including both diffuse and focal brain injury as a result of the initial SAH as well as subsequent complications, and thus different treatments and pathways of care, response to rehabilitation and functional outcomes can be anticipated [6]. Furthermore, non-traumatic SAH is excluded from Australian Clinical Guidelines for Stroke Management [3] due to the differing pathophysiology to that of ischemic stroke or intracerebral

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hemorrhage. Guidelines for aneurysmal SAH management exist in the USA [8]; however, specific recommendations for dysphagia management in this population do not currently exist.

Dysphagia (difficulty swallowing) is a common sequela of stroke, with its consequences well described in the literature, for e.g., pneumonia, malnutrition, dehydration, and asphyxiation [9–11]. Increased hospital length of stay and significantly higher healthcare costs associated with dysphagia following stroke and acute hospital admission have been well described [12–14]. However, dysphagia following non-traumatic SAH has received relatively little attention in systematic studies.

Dysphagia may arise as a primary consequence of the SAH or secondary to complications associated with treatment, such as intubation or surgical intervention [15–18]. Studies investigating dysphagia following non-traumatic SAH have identified high failure rates during dysphagia screening between 29 and 77.7% [19–22]. On videofluoroscopic swallowing study (VFSS), more than 50% of participants presented with both oral and pharyngeal phase deficits at 33.7(± 11.1) days post onset of aneurysmal SAH [15]. Risk factors for dysphagia that persisted at 6 months following onset were initial Glasgow Coma Scale (GCS) score, hemorrhage volume, presence of intraventricular hemorrhage, and degree of cognitive deficits [15]. Patients with more severe SAH (WFNS grade 3–5) are more likely to require rehabilitation for eating and swallowing function than those with less severe SAH [23]. In a rehabilitation context, 17.9% of individuals presented with significant dysphagia requiring tube feeding [24]. Dysphagia is expected to persist at 6 months post onset in up to 26.6% of individuals following non-traumatic SAH [19]. Dysphagia as a consequence of surgical clipping or endovascular coiling has also been reported in single case studies [16, 17, 25].

Despite this knowledge, previous studies have relied on brief screening tools which were not specific to individuals following non-traumatic SAH [20–22], or reliance on particular clinical indicators (e.g., pneumonia rates) to determine dysphagia incidence in the acute phase [19, 22]. To date, no studies have systematically explored dysphagia incidence and risk factors for individuals within the acute phase following non-traumatic SAH. Therefore, SLPs currently have limited data to guide their understanding of individuals most at risk of dysphagia within the acute phase following non-traumatic SAH. This lack of knowledge has the potential to impact health outcomes, and influence decisions on care and resource provision for this patient group. This study aims to investigate the incidence of dysphagia in a retrospective cohort of patients following non-traumatic SAH. Additionally, this study seeks to ascertain risk factors that may contribute to increased

dysphagia risk. It is anticipated that establishment of incidence and risk factors for dysphagia will support early identification and referral to SLP services.

## Methods

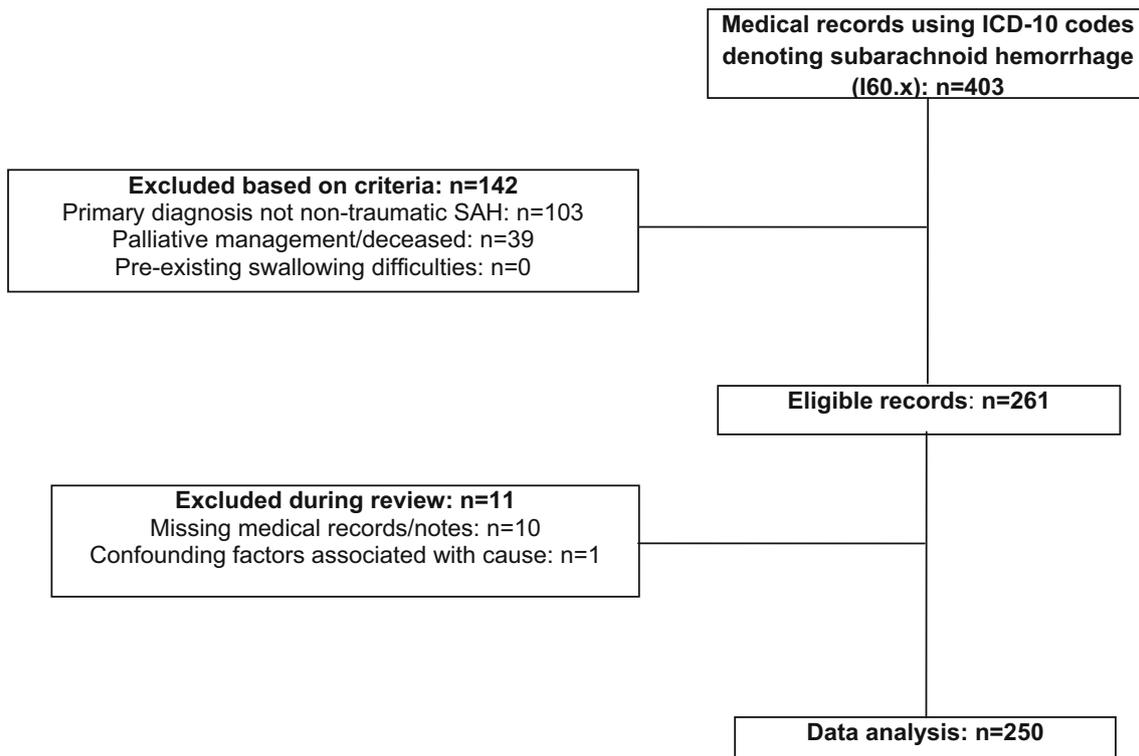
### Study Design and Population

A retrospective chart review of the medical records of adults ( $\geq 18$  years of age) who were admitted to the Royal Brisbane and Women's Hospital (RBWH) with acute, primary, non-traumatic SAH between January 2013 and December 2015 (3-year period) was conducted. The RBWH's 'Kenneth G. Jamieson Neurosurgery Unit' is the major neurosurgery referral center for Queensland. The World Health Organization ICD-10 diagnostic code 'I60 – subarachnoid hemorrhage' was used to identify all appropriate cases where SAH was diagnosed during admission. A total of 403 cases were identified. Adults with a primary diagnosis that was not non-traumatic SAH ( $n = 103$ ), those who received only palliative management or died during their admission secondary to complications from their injuries ( $n = 39$ ), and adults with pre-existing swallowing issues ( $n = 0$ ) were excluded based on pre-determined eligibility criteria. A further ten participants were excluded due to missing medical records or incomplete admission notes, and one participant was excluded for the presence of confounding factors associated with cause upon review of their medical chart. A total of 250 participants were eligible for inclusion in the data analysis (Fig. 1).

Participants were 151 females and 99 males with a mean age of 54.73 (SD = 12.9; range = 18–92) years. The majority of participants were previously independent ( $n = 245$ , 98%) with no reported impairments in cognition ( $n = 238$ , 95.2%) prior to their hospital admission. Aneurysms were the most common cause of non-traumatic SAH ( $n = 185$ ), with 15.68% ( $n = 29/185$ ) of these participants having multiple aneurysms identified on diagnostic imaging. The total mean length of hospital stay was 19.81 days (SD = 14.63, range = 0–136). Table 1 further outlines the demographic details of the total population.

### Data Collection

A uniform data abstraction tool, developed based on the logical flow of existing medical records, was used to enhance internal validity and reproducibility of the study [26]. A second SLP reviewed a selection of the medical charts to ensure consistency of data collection and to ensure inter-rater reliability [27]. Data were collected from the point of initial hospital admission until discharge from their acute hospital stay.



**Fig. 1** Flow diagram outlining eligibility criteria

Clinical parameters relating to participant demographics (age, gender), medical history, social history, and pre-admission cognitive status were collected. Pre-admission cognitive status was reported by a health professional in the participants' medical chart following caregiver reports. Information related to the non-traumatic SAH, including initial clinical presentation, date of onset, date of hospital admission, severity, cause, location, date of diagnostic studies, interventional procedures (e.g., craniotomy and clipping), intensive care unit (ICU) admission dates, intubation and ventilation dates and details, tracheostomy details, medical complications during admission, presence of multiple aneurysms, and discharge date and destination was also collected. Participants were determined to have had a 'new stroke' as a primary complication if they developed a new ischemic event during the admission.

Although non-traumatic SAH severity is universally rated using a variety of scales, the most common being WFNS [28] and Fisher scale [29], only WFNS grade was extracted for inclusion in this study as a larger proportion of participants had WFNS scores reported ( $n = 221$ ) compared with Fisher scale scores ( $n = 210$ ). The WFNS system considers both an individual's GCS score and presence of focal neurological deficits (e.g., hemiparesis or hemiplegia and/or aphasia) to determine the severity of the injury and assist prediction of patient outcomes and

prognosis [28]. Meanwhile, the Fisher grading scale determines the amount and distribution of subarachnoid blood and assigns a score accordingly [29]. The WFNS grade can be determined via clinical patient assessment whilst grading via the Fisher scale requires radiological assessment using non-contrast CT head scanning.

Participants were considered to have dysphagia if they demonstrated difficulties in the oral and/or pharyngeal phases of swallowing on clinical swallowing examination conducted by a SLP or if swallowing concerns were documented in the medical chart from a medical or nursing staff member which may or may not have led to the prescription of modified diet and/or fluids. The research team employed a binary rating of dysphagia or no dysphagia based on these reports. SLP assessment occurred upon referral from another health professional when the patient was deemed medically stable. Medical stability was defined as a stable respiratory system, ability to tolerate an upright position, and ability to maintain sufficient level of consciousness for assessment. A number of SLPs were involved in the care of these participants across the 3 years. Clinical swallowing examination practices were performed and recorded in accordance with Speech Pathology Australia's dysphagia clinical guidelines [30].

**Table 1** Demographics of patients with non-traumatic subarachnoid hemorrhage

Variable	Result
Pre-admission cognitive status	
Nil cognitive impairment	95.20% ( <i>n</i> = 238)
Possible cognitive impairment	3.60% ( <i>n</i> = 9)
Confirmed cognitive impairment	1.20% ( <i>n</i> = 3)
Social history	
Previously independent	98.00% ( <i>n</i> = 245)
Independent with ADLs, assistance with IADLs	1.20% ( <i>n</i> = 3)
Assistance with all ADLs	0.80% ( <i>n</i> = 2)
WFNS grade	
1	54.8% ( <i>n</i> = 137)
2	12% ( <i>n</i> = 30)
3	3.68% ( <i>n</i> = 9)
4	9.6 ( <i>n</i> = 24)
5	8.4% ( <i>n</i> = 21)
Not reported	11.6% ( <i>n</i> = 29)
Cause	
Aneurysm	73.6% ( <i>n</i> = 184)
AVM	1.69% ( <i>n</i> = 4)
Aneurysm and AVM	0.40% ( <i>n</i> = 1)
DAVF	1.20% ( <i>n</i> = 3)
Perimesencephalic	4.00% ( <i>n</i> = 10)
Cerebral venous sinus thrombosis	0.40% ( <i>n</i> = 1)
Drug associated	0.80% ( <i>n</i> = 2)
Not identified	18.00% ( <i>n</i> = 45)
LOHS (days)	<i>M</i> = 19.81 ( <i>SD</i> = 14.63) Range 0–136

*M* mean, *SD* standard deviation, *ADLs* activities of daily living, *IADLs* instrumental activities of daily living, *LOHS* length of hospital stay, *AVM* arteriovenous malformation, *DAVF* dural arteriovenous fistula

## Data Analysis

All collected data were entered into a Microsoft Excel spreadsheet. Descriptive statistics were used to establish mean, standard deviation, range, and percentage for participant demographic variables and dysphagia incidence. To allow for data analysis using a binary outcome, WFNS grading scores were grouped into ‘good grade’ and ‘poor grade’, consistent with Karic et al. [23]. ‘Good grade’ was assigned to participants with WFNS grade of 1 or 2 (*n* = 167; motor deficits absent and GCS  $\geq$  13), whilst ‘poor grade’ was assigned to participants with WFNS 3, 4, or 5 (*n* = 54; motor deficits present and GCS 13–14 or motor deficits present or absent with GCS  $\leq$  12). Interventional procedure type was analyzed if it was related to the primary treatment of the non-traumatic SAH (e.g., craniotomy and clipping, endovascular coiling). Stata 13 (StataCorp, College Station, Texas) was used to perform statistical analysis.  $\chi^2$  and Fisher’s exact tests for categorical data, and unpaired *t* tests for continuous data were

conducted to compare the characteristics of individuals with and without dysphagia for potential predictive power of dysphagia. Due to the retrospective nature of this study, data for all variables were not available. Where sufficient detail was lacking or data were unreported or not relevant to a participant, a percentage of ‘unknown’ is reported. Logistic regression analysis was performed to evaluate predictive factors with positive report of dysphagia as the primary outcome variable. A *p* value of  $< 0.01$  was used to indicate statistical significance due to multiplicity of tests [31].

The current study received ethical clearance from the RBWH and The University of Queensland Human Research Ethics Committees. A Public Health Act application was also approved for the release of confidential health information (medical records) without the participant’s consent.

## Results

### Incidence of Dysphagia Following Non-traumatic Subarachnoid Hemorrhage

The incidence of dysphagia was 31.6% ( $n = 79$ ) over the three-year period. Dysphagia was diagnosed by SLPs for 92.41% ( $n = 73/79$ ) of participants; 84.81% ( $n = 67/79$ ) via clinical swallowing examination (CSE) alone; and 7.59% ( $n = 6/79$ ) through a combination of CSE and instrumental swallowing assessment (VFSS or fiberoptic endoscopic evaluation of swallowing). The remaining 7.59% ( $n = 6$ ) of participants with dysphagia had their dysphagia identified and managed by medical or nursing staff, and were not seen by SLP during their acute hospital admission.

### Characteristics of the Population With and Without Dysphagia

In order to explore what variables may predict dysphagia outcome following non-traumatic SAH,  $\chi^2$ , Fisher's exact, and  $t$  tests were used to compare dysphagic and non-dysphagic groups. The average length of hospital stay was significantly longer ( $p < 0.01$ ) in participants with dysphagia (31.71 days,  $SD = 18.83$ , range = 3–136) compared to those without dysphagia (14.31 days,  $SD = 7.38$ , range = 0–48). There were no significant differences between the two groups based on gender, presence of multiple aneurysms, social history, pre-admission cognitive status, aneurysm location, side of brain, circulation, re-bleeding, or date from SAH onset to admission. Analysis revealed statistically significant differences ( $p < 0.01$ ) for all other variables (see Table 2).

### Establishment of Predictive Factors for Dysphagia Following Non-traumatic Subarachnoid Hemorrhage

To determine the set of variables that could be used to reliably predict dysphagia outcome following non-traumatic SAH via multivariate logistic regression modeling, the statistical relationship between dysphagia outcome and the variables identified as being significantly different between groups were each investigated using a univariate logistic regression model (Table 3). Tracheostomy was excluded from further analysis as all participants in the cohort who required tracheostomy ( $n = 10$ ) presented with dysphagia. For the remaining variables, sensitivity (i.e., correct identification of patients with dysphagia), ranged from 0% (need for ICU admission, need for interventional procedure, hydrocephalus, type of primary interventional

procedure) to 82.28% for need for intubation and ventilation, and number of intubations. Specificity (i.e., correct identification of patients without dysphagia) was high for all variables, ranging between 78.95% for number of interventional procedures and 100% for need for ICU admission, need for interventional procedure, and hydrocephalus. Positive predictive value (PPV), or the proportion of participants with the variable present that would be correctly diagnosed with dysphagia, ranged from 0% for type of primary interventional procedure to 82.28% for length of intubation. PPV for need for ICU admission, need for interventional procedure, hydrocephalus, type of primary interventional procedure, and cause were undefined as sensitivity was 0% for these variables. These variables were thus excluded from use in the multivariate regression model. The negative predictive value (NPV), or the proportion of participants without the variable present that would be correctly diagnosed with dysphagia, ranged from 68.40% for need for ICU admission, need for interventional procedure, and hydrocephalus, to 91.08% for need for intubation and ventilation, and number of intubations.

Despite a statistically significant difference in the number of ICU admissions between individuals with ( $M = 1.19$ ) and without dysphagia ( $M = 0.95$ ), this difference was not considered a clinically significant variable. All individuals had an average of 1 day admitted to ICU and therefore this variable was excluded from further analysis.

Need for intubation and ventilation, number of intubations, WFNS grading, and number of intubations were excluded from use in the multivariate regression model due to high odds ratios. Examination of the remaining variables for multicollinearity revealed variation inflation factors  $< 10$ , and therefore the remaining variables (age, vasospasm, new stroke, ICU LOS, and length of intubation) were included in the final regression model. Although unsuitable for use in multivariate regression modeling, data diagnostics revealed significant relationships between these variables and dysphagia presence. The odds of developing dysphagia in individuals who needed to be intubated and ventilated were 23.71 times higher than in those who did not need to be intubated and ventilated. The odds of presenting with dysphagia increased by 11.48 times for individuals with WFNS 3–5 compared with WFNS 1–2. For each additional day intubated, the odds of presenting with dysphagia increased 24.76 times. For each additional day of intubation, the odds of presenting with dysphagia increased by 10.8 times.

Five remaining variables were deemed suitable for inclusion in multivariate regression modeling: age, ICU LOS, vasospasm, new stroke, and length of intubation. Vasospasm and new stroke did not achieve statistical significance in the model. The model was re-run, removing

**Table 2** Between-groups comparison of variables hypothesized to be related to dysphagia risk in patients with and without dysphagia following non-traumatic subarachnoid hemorrhage

Population variable	Dysphagic ( <i>n</i> = 79)		Non-dysphagic ( <i>n</i> = 171)		<i>p</i> value*	
	<i>n</i>	%	<i>n</i>	%		
Gender						
Male	36	45.57	63	36.84	0.19	
Female	43	54.43	108	63.16		
WFNS grade ( <i>n</i> = 221)						
1	25	31.65	112	65.5	< 0.01	
2	11	13.92	19	11.11		
3	6	7.59	3	1.75		
4	18	22.78	6	3.51		
5	17	21.52	4	2.34		
Cause						
Aneurysm	72	91.14	112	65.5	< 0.01	
Arteriovenous malformation	0	0	4	2.34		
Aneurysm and arteriovenous malformation	1	1.27	0	0		
Dural arteriovenous fistula	1	1.27	2	1.17		
Perimesencephalic	0	0	10	5.85		
Cerebral venous sinus thrombosis	1	1.27	0	0		
Drug associated	0	0	2	1.17		
Not identified	4	5.06	41	23.98		
Multiple aneurysms	8	10.12	21	12.28		0.154
Need for ICU admission	77	97.47	147	85.96		< 0.01
Need for interventional procedure	75	94.94	115	67.25	< 0.01	
Primary interventional procedure type						
None	7	8.86	59	34.50	< 0.01	
Surgical craniotomy and clipping	24	30.38	29	16.96		
DSA and coiling	48	60.76	82	47.95		
Embolization	0	0	1	0.58		
Need for intubation and ventilation	65	82.28	28	16.37	< 0.01	
Tracheostomy	10	12.66	0	0	< 0.01	
Primary complications						
Vasospasm	35	44.30	30	17.54	< 0.01	
Hydrocephalus	40	50.63	42	24.56	< 0.01	
Re-bleeding	3	3.80	0	0	0.031	
New stroke	15	18.99	7	4.09	< 0.01	
	Mean ± SD	Range	Mean ± SD	Range	<i>p</i> value*	
Age (years)	58.33 ± 12.93	26 to 92	53.07 ± 12.58	18 to 83	< 0.01	
ICU LOS (days)	14.01 ± 6.87	0 to 38	5.25 ± 4.58	0 to 20	< 0.01	
Number of ICU admissions	1.19 ± 0.45	0 to ≥ 2	0.95 ± 0.48	0 to ≥ 2	< 0.01	
Number of intubations	1.22 ± 0.87	0 to 4	0.19 ± 0.45	0 to 2	< 0.01	
Length of intubation (days)	5.34 ± 4.96	0 to 19	0.33 ± 0.89	0 to 6	< 0.01	

ICU LOS Intensive Care Unit length of stay, DSA digital subtraction angiography, WFNS World Federation of Neurological Surgeons

\**p* values based on  $\chi^2$ , fisher's exact, and *t* tests

each variable individually and both variables together, until significance for all individual variables, and the regression model was obtained. The three variables included in the final model were age, ICU LOS, and length of intubation.

The results, outlined in Table 4, indicate that the strongest predictor of developing dysphagia following non-traumatic SAH is length of intubation. Individuals are 2.09 times more likely to develop dysphagia for every additional

**Table 3** Variables for dysphagia risk

Variable	SENS (%)	SPEC (%)	PPV (%)	NPV (%)	+LR	-LR	CC
Need for ICU admission	0	100	UD	68.40	0	1	68.40
Need for interventional procedure	0	100	UD	68.40	0	1	68.40
Need for intubation and ventilation	82.28	83.63	69.89	91.08	4.82	0.22	83.20
Hydrocephalus	0	100	UD	68.40	0	1	68.40
Vasospasm	44.30	82.46	53.85	76.22	2.44	0.68	70.40
New ischaemic event	18.99	95.91	68.18	71.93	11	0.84	71.60
Age	3.80	98.83	60	68.98	4	0.97	68.80
Type of primary interventional procedure	0	99.42	0	68.27	0	1.01	68
Number of interventional procedures	73.42	78.95	61.70	86.54	3.76	0.34	77.20
ICU LOS	63.29	87.72	70.42	83.80	5.25	0.42	80
Number of ICU admissions	21.52	91.23	53.13	71.56	2.33	0.86	69.20
Number of intubations	82.28	83.63	69.89	91.08	5.13	0.21	83.20
Length of intubation	70.65	91.14	82.28	84.21	7.89	3.22	83.60
WFNS grading	53.25	90.97	75.93	78.44	5.89	0.52	77.83
Cause	0	100	UD	68.40	0	1	68.40

SENS sensitivity, SPEC specificity, PPV positive predictive value, NPV negative predictive value, +LR positive likelihood ratio, -LR negative likelihood ratio, CC correctly classified, UD undefined

day intubated. In addition, individuals with non-traumatic SAH are 1.06 times more likely to develop dysphagia for every year increase in their age and are 1.21 times more likely to develop dysphagia for every additional day they stay in ICU.

The Hosmer–Lemeshow (H–L) goodness-of-fit test and receiver-operating characteristic (ROC) analysis was performed to evaluate the goodness-of-fit of the multivariate regression model data [32, 33]. The H–L statistic was non-significant ( $p = 0.6315$ ) and the area under the ROC curve was non-significant ( $p = 0.9297$ ), both indicating that the model's goodness-of-fit was appropriate and highly accurate.

The model's overall sensitivity was 72.15% and specificity was 94.15%. Therefore, using the variables age, ICU LOS, and length of intubation, over 7 out of every 10 patients would be correctly identified with dysphagia using this model. The model would also correctly identify individuals without dysphagia in over 9 out of every 10 individuals. PPV of the model in accurately predicting dysphagia was 85.07% whilst the NPV in predicting no dysphagia was 87.98%. The likelihood that a clinician would predict dysphagia is 12 times greater than an individual who does not have these characteristics. It is 0.29

times less likely that an individual with dysphagia is identified than an individual who does not have these characteristics. Overall, 87.20% of individuals would be correctly classified using this model.

### Final Criteria Set

To enable a clinical application of definitive cut-points for continuous variables (age, ICU LOS, and length of intubation) that may aid in dysphagia identification, ROC analysis and the Youden index ( $J$ ) was completed. ROC analysis takes into account the epidemiologic situation (e.g., incidence) as well as the relative consequences of false-positive and false-negative results whilst optimizing the cut-off value [32]. The Youden Index ( $J$ ), a function of sensitivity and specificity where sensitivity and specificity are optimized and equally weighted, was used to investigate overall diagnostic effectiveness [34–38]. Economically, equal sensitivity and specificity and thus estimating the cut-point and  $J$  is considered a cost-effective and statistically sound approach [39].

The optimal cut-off points identified were:

- 57.5 years for age. Sensitivity was 56%; more than five out of every ten individuals with dysphagia following

**Table 4** Final multivariate regression model

Variable	Odds ratio (adjusted)	$p$ value	95% confidence interval	
Age	1.06	0.001	1.02	1.10
ICU LOS	1.21	0.000	1.10	1.33
Length of intubation	2.09	0.000	1.58	2.76

non-traumatic SAH would be correctly identified using age alone. Specificity was 64%; approximately six out of every ten individuals without dysphagia following non-traumatic SAH would be correctly identified using age alone.

- 7.5 days for ICU LOS. Sensitivity was 91%; more than nine out of every ten individuals with dysphagia following non-traumatic SAH would be correctly identified using ICU LOS. Specificity was 68%; more than six out of every ten individuals without dysphagia following non-traumatic SAH would be correctly identified using ICU LOS alone.
- 1.5 days for length of intubation. Sensitivity was 77%; more than seven out of every ten individuals with dysphagia following non-traumatic SAH would be correctly identified using length of intubation. Specificity was 90%; nine out of every ten individuals without dysphagia following non-traumatic SAH would be correctly identified using length of intubation.

The series of analyses above identified the following set of clinical criteria that, in combination or isolation, could be used to predict an individual's risk of dysphagia following non-traumatic SAH. These criteria included age > 57.5 years, ICU LOS > 7.5 days, length of intubation > 1.5 days, need for tracheostomy, vasospasm, and new stroke.

When retrospectively applied to the current cohort, sensitivity of the criteria set was 100%, and specificity was 41.52% (see Table 5). Therefore, when using these criteria as part of a screening test for dysphagia, all individuals presenting with dysphagia after non-traumatic SAH would be correctly identified. However, the criteria set over-identifies individuals who may be at risk of dysphagia (PPV 44.13%) and who require screening. Reassuringly, individuals who obtain a negative result using the criteria set should not have dysphagia (NPV 100%).

**Table 5** Contingency table of the final criteria set for dysphagia risk following non-traumatic subarachnoid hemorrhage

Test outcome	Dysphagia status	
	Positive	Negative
Positive	79 ( <i>a</i> = true positive)	100 ( <i>b</i> = false positive)
Negative	0 ( <i>c</i> = false negative)	71 ( <i>d</i> = true negative)

$$\text{Sensitivity} = a/(a + c) = 79/(79 + 0) = 100\%$$

$$\text{Specificity} = d/(b + d) = 71/(100 + 71) = 41.52\%$$

$$\text{PPV} = a/(a + b) = 79/(79 + 100) = 44.13\%$$

$$\text{NPV} = d/(c + d) = 71/(0 + 71) = 100\%$$

## Discussion

The primary aim of this study was to investigate dysphagia incidence and establish risk factors that may predict dysphagia in a retrospective cohort of individuals in the acute phase following non-traumatic SAH. The incidence rate of dysphagia identified in this study was 31.6%. From an Australian perspective, there is currently no other comparable figure available. A number of studies have examined failed dysphagia screening rates in stroke populations. A large-scale retrospective study from the USA of 18,017 individuals post stroke examining dysphagia screening results, reported a 29.63% ( $n = 254/857$ ) failure rate after SAH [20]. As this study did not specify whether the SAH cohort included both traumatic and non-traumatic causes, this statistic should be interpreted with caution with respect to non-traumatic SAH alone. Due to the nature of the injury, individuals with traumatic SAH also typically present with subdural hemorrhage, skull fracture, and traumatic contusions in the context of multiple injuries [40]. Isolated traumatic SAH is uncommon with lower overall injury severity, and would rarely necessitate speech pathology input [41]. Conversely, the high incidence rate identified in the current study supports the need for timely referral for swallowing assessment by SLPs within the acute context for individuals admitted with non-traumatic SAH. Furthermore, this figure provides an insight into the potential demands on the multidisciplinary team in relation to dysphagia management and rehabilitation of the adult patient with non-traumatic SAH which may guide resource allocation.

To enable prompt identification of individuals at risk of dysphagia and battle ever-increasing time, personnel, and economic shortages, the use of screening tools designed to classify individuals upon hospital admission have become increasingly necessary in acute care settings [42–44]. These tools can be carried out with minimal cost and time, and are non-invasive with minimal risk to the patient [44, 45]. Timely assessment and management of dysphagia in acute stroke populations has been shown to improve patient outcomes (e.g., reduced respiratory complications and length of stay), and thus reduce the overall cost to the health system [46–48]. As a result, there is support for the development of formal dysphagia screening tools that encompass evidence-based, etiology-specific criteria for dysphagia risk and can be conducted by other members of the healthcare team, such as the Yale Swallow Protocol [42, 44, 49]. Therefore, the second aim of this study was to establish a set of potential predictive factors which may predict dysphagia risk in individuals following non-traumatic SAH during the acute admission. The establishment of tailored risk factors for individuals with non-traumatic

SAH will ensure that members of the multidisciplinary team can appropriately identify individuals who are at risk of dysphagia and would benefit from referral to SLP services, and prevent associated complications.

The final core set of risk factors for dysphagia following non-traumatic SAH identified in the current study included age > 55.5 years, ICU LOS > 7.5 days, length of intubation > 1.5 days, need for tracheostomy, vasospasm, and new stroke. A number of the identified risk factors such as age [50, 51], tracheostomy [52], intubation and ventilation [18], ICU admission [18], and stroke [10] are known to influence dysphagia risk during hospitalization. However, the current study provides further validation of the importance of these criteria in the non-traumatic SAH population. Furthermore, additional factors that independently contribute to dysphagia risk, increasing the sensitivity of the overall criteria set, have been identified. For example, individuals who developed vasospasm were also at higher risk of developing dysphagia.

Retrospective application of the criteria set to this study's cohort identified the sensitivity of the non-traumatic SAH-specific dysphagia risk which was high at 100%, with a specificity of 41.52%. The risk criteria have a high sensitivity, and thus all individuals with dysphagia should be identified using the risk criteria set alone. If the risk criteria are not met, the clinician can be sure that there is no dysphagia in all individuals (NPV = 100%). For economic viability, equal sensitivity and specificity are desirable so that there is minimization of unnecessary full screening assessments and SLP referrals. Even though a lower specificity may lead to over-identification of individuals requiring a full dysphagia screen, a lower specificity assures that all individuals with dysphagia risk are identified and are thus able to receive appropriate referral, assessment, and management. A lower specificity for dysphagia screening has been accepted in the stroke population with studies identifying a high proportion of individuals requiring SLP assessment (68–77.7%) for dysphagia based on screening tests alone [21, 22].

Despite this study providing promising insights into dysphagia incidence and potential risk factors following non-traumatic SAH, there are a number of limitations in this study related to its retrospective design. Patients admitted with non-traumatic SAH were not all routinely screened for dysphagia or assessed by SLPs to have dysphagia status confirmed, and therefore the incidence rate reported in this study may not be a true accurate representation. Multiple SLPs were involved in patient care of this population over the 3-year recruitment period, and thus SLP assessment practices may have been variable in accurately identifying the nature or severity of dysphagia. A prospective study with patients following non-traumatic SAH is currently underway with patient assessments being

conducted by one SLP. It is acknowledged that CSE alone does not provide a reliable indication of true dysphagia incidence. However, the use of CSE as the primary diagnostic tool in this study, aligns with common SLP practice in Australia [53]. Future studies should incorporate instrumental evaluation of swallowing with this patient cohort to more accurately define the nature and incidence of dysphagia following non-traumatic SAH. The inclusion of patients with medical and/or nursing staff reports of dysphagia ( $n = 6$ ) may have also impacted dysphagia incidence findings. The variables able to be explored in this study were also limited by the information that is routinely recorded by the multidisciplinary team in participants' medical charts. Data for all variables were not available. Other studies have identified the presence of intraventricular hemorrhage, initial GCS score, hemorrhage volume, and degree of cognitive deficits as risk factors for chronic dysphagia [15]; however, these variables were not explored as part of the current study.

Establishing risk criteria will support the timely identification of individuals at risk of developing dysphagia following non-traumatic SAH by members of the multidisciplinary hospital team. Whilst this research has established a set of clinical criteria for determining dysphagia risk specific to non-traumatic SAH, this final set of risk criteria requires validation using a prospective patient cohort. In the development of a screening instrument based on risk criteria, it is essential to consider the feasibility and training needs of medical and nursing staff who will likely be required to administer the dysphagia screening tools with patients. Prospective evaluation of the reliability and validity of the criteria set, as well as the process of service implementation, in a major metropolitan hospital in Australia has commenced in 2018. Furthermore, additional variables previously established to be related to dysphagia in non-traumatic SAH, as stated above, will be explored. It is hoped that the utilization of an evidence-based dysphagia screening procedure that contains criteria specific to the non-traumatic SAH population will aid the multidisciplinary team in promptly identifying those who need referral to SLP, potentially reducing medical and economic costs associated with late diagnosis and treatment of dysphagia.

## Conclusion

This study provides preliminary information regarding dysphagia incidence and risk factors within the acute phase following non-traumatic SAH. Dysphagia is highly prevalent in this population, and early intervention is considered critical to prevent associated complications, such as aspiration pneumonia. The set of risk factors identified will

enhance awareness of those individuals most at risk of dysphagia, and encourage members of the multidisciplinary team to identify dysphagia early in the acute phase and prompt timely referral to SLP services.

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## Compliance with Ethical Standards

**Conflict of interest** The authors declare that they have no conflict of interest.

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