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A bitter pill to swallow: dysphagia in cervical spine injury

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ABSTRACT

Background: Dysphagia is a common complication after cervical spine trauma with spinal cord injury. We sought to characterize the prevalence of dysphagia within a total cervical spinal injury (CSI) population, considering the implications of spinal cord injury status and age on dysphagia development. We hypothesized that while greater rates of dysphagia would be found in geriatric and spinal cord–injured subgroups, all patients presenting with CSI would be at heightened risk for swallowing dysfunction.

Methods: All trauma admissions to a level II trauma center from January 2010 to April 2014 with CSI were retrospectively reviewed. CSI was classified as any ligamentous or cervical spinous fracture with or without cord injury. Patients failing a formal swallow evaluation were considered dysphagic. The implications of dysphagia development on age and spinal cord injury status were assessed in univariate and multivariate analyses.

Results: A total of 481 patients met study inclusion criteria, of which 123 (26%) developed dysphagia. Within the dysphagic subpopulation, 90 patients (73%) were geriatric, and 23 (19%) sustained spinal cord injury. The dysphagic subpopulation was predominantly free from spinal cord injury (81%). Multivariate analyses found age (adjusted odds ratio: 1.06; 95% confidence interval 1.04–1.07; $P < 0.001$) and spinal cord injury (adjusted odds ratio: 2.69; 95% confidence interval 1.30–5.56; $P = 0.008$) to be significant predictors of dysphagia development.

Conclusions: Despite spinal cord–injured patients being at increased risk for dysphagia, most of the dysphagic subpopulation was free from spinal cord injury. Geriatric and CSI patients with or without cord injury should be at heightened suspicion for dysphagia development.

1. Background

The association between traumatic cervical spinal cord injury and the development of dysphagia, or swallowing difficulty, has been well established in the literature [1–9]. Previous studies investigating the prevalence of swallowing dysfunction within this population have reported rates of dysphagia ranging from 17% to 41% [1–4]. Despite a plethora of investigations detailing the impact of spinal cord damage on dysphagia development, no analyses, in review of available

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literature, have broadened their study population to include nonspinal cord–affected cervical spinal injuries (CSIs). Considering the location and traumatic mechanism of these injuries, it is feasible to suggest patients afflicted by nonspinal cord–involved CSIs could also be at increased risk for swallowing dysfunction. Due to the fact that dysphagia can lead to secondary complications including aspiration [10–12] and hospital-acquired pneumonia [13,14], identifying predictors and patients at risk for developing this swallowing abnormality is critical.

Although spinal cord damage is one of the most supported risk factors for dysphagia development [1–9], other predictors including ventilator use [1,15], tracheostomy [1,3,15–17], and halo vest fixation [18–20] are gaining added traction in the literature, particularly among the traumatically injured [1,3,15,20]. In addition, age, although already a well-established predictor for nontraumatic dysphagia development [21–26], is also emerging as a potential risk factor within this population. [1,3].

The purpose of this study was to investigate the prevalence of clinically diagnosed dysphagia within a total CSI population (both spinal cord–injured and nonspinal cord–injured subgroups), while examining the impact of age, spinal injury status, and other predictors on dysphagia development. We hypothesized that while greater rates of dysphagia would be observed in geriatric and spinal cord–injured subgroups, all patients presenting with CSI would be at heightened risk for swallowing dysfunction.

2. Methods

After study review and approval by the organization's institutional review board, the trauma registry of the Pennsylvania Trauma Systems Foundation (Digital Innovations, Forest Hill, MD) was retrospectively queried for all trauma admissions presenting with CSI to an accredited level II community trauma center from January 2010 to April 2014. CSI was defined as any ligamentous injury or cervical spinous fracture with or without damage to the spinal cord. Demographic variables of interest included age, gender, Injury Severity Score (ISS), Glasgow Coma Scale (GCS) score, traumatic brain injury (TBI), facial fracture, past medical history of stroke, vertebral injury location (cervical vertebrae number [C1–C7]), number of vertebrae injured, and spinal cord injury. TBI was defined as an insult/injury to the brain resulting in a positive head computed tomography scan, and/or a diffuse axonal injury (including concussion with or without a loss of consciousness). Outcome variables collected included hospital length of stay (LOS), ventilator use, tracheostomy, halo vest fixation, and dysphagia. To determine whether patients in the study population developed dysphagia, interrogation of individual medical records in the Epic electronic medical record system (Epic Systems Corporation, Verona, WI) was conducted. Patients with suspicion of swallowing dysfunction after bedside nursing assessment who subsequently failed a formal swallow evaluation by speech pathology were considered clinically dysphagic. It should be noted that per our practice protocol, swallowing dysfunction is not tested in patients with a tracheostomy until after it has been removed. Patients discharged, transferred, or dying before receiving in-hospital oral intake were excluded from analysis as to limit our study population to CSI patients definitively able to be classified as dysphagic or non-dysphagic.

2.1. Data analysis

Data analysis was conducted using Stata 14 statistical analysis software (StataCorp 2015). Continuous variables were represented as means and standard deviations and categorical variables as counts and percentages. To analyze the implications of collected variables on dysphagia development, Pearson χ^2 analysis was performed. To evaluate the impact of age on dysphagia development, the total study population was divided into geriatric ($\geq$65 y old) and general ($<$65 y old) subgroups. Similarly, comparisons between spinal cord–injured and nonspinal cord–injured subgroups were also conducted.

Univariate logistic regression models were subsequently used to calculate unadjusted odds ratios (ORs) of dysphagia development for age, gender, ISS, GCS, TBI, facial fracture, past medical history of stroke, spinal cord injury, hospital LOS, ventilator use, tracheostomy, and halo vest fixation. A multivariate binary logistic regression model of dysphagia was then used to calculate adjusted ORs (AOR) for variables found to be significant predictors of dysphagia in univariate analysis. To determine the discrimination of the multivariate model, the area under the receiver operating characteristic was calculated and graphed. A P value <0.05 was considered statistically significant.

3. Results

Over the 4-y study period, 546 trauma patients presented with CSI. Of this, 65 patients were excluded from analysis for being discharged, transferred, or dying before receiving oral intake, producing a final eligible population of 481 CSI patients. The study population was predominantly composed of white (97%), male (56%) trauma patients aged less than 65 y (53%). An overview of demographic variables for the study population is summarized in Table 1. The primary mechanisms of injury were falls (54%), followed by motor vehicle collisions (36%). A total of 272 patients (57%) suffered vertebral fracture/ligamentous injury to only one cervical vertebrae, whereas 209 (43%) sustained injury to multiple cervical vertebrae. The most common injury occurred at the level of C2 (34%), followed by C6 (29%). Within the total CSI study population, 56 patients (12%) presented with spinal cord injury in addition to their fracture/ligamentous injury.

Of the 481 CSI cases under investigation, 123 (26%) patients developed dysphagia. Within this dysphagic subpopulation, 90 patients (73%) were aged $\geq$65 y, and 23 (19%) sustained a spinal cord injury. Pearson χ^2 analysis found a significantly higher rate of dysphagia in the geriatric population compared with the general population (90/227 [40%] *versus* 33/254 [13%]; $P < 0.001$) and in spinal cord–injured patients compared with nonspinal cord–injured patients (23/56 [41%] *versus* 100/425 [24%]; $P = 0.018$; Table 1). Furthermore, univariate regression analyses found age (OR: 1.03; 95% CI 1.02–1.04; $P < 0.001$) and spinal cord injury (OR: 2.41; 95% CI

Table 1 – Demographics: total study population, dysphagic and non-dysphagic subpopulations.

Total CSI study population ($n = 481$)		Dysphagic ($n = 123$)	Non-dysphagic ($n = 358$)	P
n (%)		*n* (%)	*n* (%)	
Age				
Mean: 59.8 ± 22.1				
General (<65)	254 (52.8)	33 (26.8)	221 (61.7)	—
Geriatric (≥65)	227 (47.2)	90 (73.2)	137 (38.3)	<0.001
Gender				
Female	211 (43.9)	55 (44.7)	156 (43.6)	—
Male	270 (56.1)	68 (55.3)	202 (56.4)	0.807
ISS				
Mean: 12.5 ± 8.89				
<12	281 (58.4)	53 (43.1)	228 (63.7)	<0.001
12–25	163 (33.9)	54 (43.9)	109 (30.5)	0.006
>25	37 (7.69)	16 (13.0)	21 (5.87)	0.019
GCS				
Mean: 14.5 ± 1.59				
≤8	10 (2.08)	7 (5.69)	3 (0.84)	0.003
9–13	25 (5.20)	9 (7.32)	16 (4.47)	0.202
14–15	446 (92.7)	107 (87.0)	339 (94.7)	0.006
TBI				
Yes	92 (19.1)	36 (29.3)	56 (15.6)	0.002
No	389 (80.9)	87 (70.7)	302 (84.4)	—
Facial fracture				
Yes	36 (7.48)	21 (17.1)	15 (4.19)	<0.001
No	445 (92.5)	102 (82.9)	343 (95.8)	—
Spinal cord injury				
Yes	56 (11.6)	23 (18.7)	33 (9.22)	0.003
No	425 (88.4)	100 (81.3)	325 (90.8)	—
Hospital LOS				
Mean: 4.97 ± 6.47				
≤2	200 (41.6)	15 (12.2)	185 (51.7)	<0.001
3–5	151 (31.4)	42 (34.2)	109 (30.5)	0.402
>5	130 (27.0)	66 (53.7)	64 (17.9)	<0.001
Ventilator				
Yes	46 (9.56)	27 (12.3)	19 (5.31)	<0.001
No	435 (90.4)	96 (87.7)	339 (94.7)	—
Tracheostomy				
Yes	14 (2.91)	9 (7.32)	5 (1.40)	0.002
No	467 (97.1)	114 (92.7)	353 (98.6)	—
Halo vest				
Yes	31 (6.45)	17 (13.8)	14 (3.91)	<0.001
No	450 (93.6)	106 (86.2)	344 (96.1)	—
History of stroke				
Yes	4 (0.83)	1 (0.81)	3 (0.84)	0.975
No	477 (99.2)	122 (99.2)	355 (99.2)	—
Dysphagia				
Yes	123 (25.6)	123 (100.0)	0 (0.00)	—
No	358 (74.4)	0 (0.00)	358 (100.0)	—

1.36–4.27; $P = 0.003$) to be significant predictors of dysphagia. In addition, ISS (OR: 1.05; 95% CI 1.03–1.08; $P < 0.001$), GCS (OR: 0.79; 95% CI 0.70–0.90; $P = 0.001$), TBI (OR: 2.17; 95% CI 1.34–3.51; $P = 0.002$), facial fracture (OR: 4.59; 95% CI 2.28–9.23; $P < 0.001$), hospital LOS (OR: 1.11; 95% CI 1.07–1.16; $P < 0.001$), ventilator use (OR: 4.94; 95% CI 2.63–9.26; $P < 0.001$), tracheostomy (OR: 5.45; 95% CI 1.79–16.6; $P = 0.003$), and halo vest fixation (OR: 3.85; 95% CI 1.84–8.06; $P = 0.001$) were found to be significantly correlated with increased ORs of dysphagia in univariate analysis (Table 2). No association was found between gender (OR: 0.95; 95% CI 0.63–1.43; $P = 0.807$), or past medical history of stroke (OR: 0.95; 95% CI 0.10–9.36; $P = 0.975$) and dysphagia development although it should be noted only four patients within the study population previously presented with a stroke.

To further investigate the impact of these covariates on dysphagia development, a multivariate logistic regression model accounting for the interaction of variables significantly associated with dysphagia in univariate analysis (age, ISS, GCS, TBI, facial fracture, spinal cord injury, hospital LOS, ventilator use, tracheostomy, and halo vest placement) was performed. Age, GCS, TBI, facial fracture, spinal cord injury, hospital LOS, ventilator use, and halo vest fixation remained significant predictors of dysphagia, whereas ISS and

Table 2 – Unadjusted and adjusted odds ratios of dysphagia development.

Variable	Unadjusted odds ratio (95% CI)	Adjusted odds ratio (95% CI)	P value
Gender (male)*	0.95 (0.63–1.43)	—	—
Stroke history*	0.95 (0.10–9.36)	—	—
Age†	1.03 (1.02–1.04)	1.06 (1.04–1.07)	<0.001
ISS‡	1.05 (1.03–1.08)	1.01 (0.98–1.04)	0.656
GCS†	0.79 (0.70–0.90)	0.81 (0.69–0.96)	0.012
TBI†	2.17 (1.34–3.51)	2.26 (1.22–4.21)	0.010
Facial fracture†	4.59 (2.28–9.23)	2.67 (1.13–6.29)	0.025
Spinal cord injury†	2.41 (1.36–4.27)	2.69 (1.30–5.56)	0.008
Hospital LOS†	1.11 (1.07–1.16)	1.04 (1.01–1.08)	0.024
Ventilator†	4.94 (2.63–9.26)	3.58 (1.41–9.11)	0.007
Tracheostomy‡	5.45 (1.79–16.6)	0.77 (0.17–3.42)	0.727
Halo†	3.85 (1.84–8.06)	6.87 (2.67–17.6)	<0.001
			AUROC = 0.83

AUROC = area under the receiver operating characteristic.
*Excluded from multivariate model—nonsignificant in univariate analysis.
†Significant in univariate and multivariate analysis.
‡Significant in univariate analysis.

tracheostomy were found insignificant (Table 2). CSI patients who underwent halo vest placement were found to have a 6.87 times greater OR of dysphagia development compared with non-halo vest counterparts (AOR: 6.87; 95% CI 2.67–17.6; $P < 0.001$). Similarly, CSI patients with spinal cord injuries were 169% more likely to develop dysphagia compared with CSI patients without spinal cord injuries (AOR: 2.69; 95% CI 1.30–5.56; $P = 0.008$). With respect to age and hospital LOS, every 1-y increase in age was associated with a 6% increase in dysphagia development (AOR: 1.06; 95% CI 1.04–1.07; $P < 0.001$), whereas every 1-d increase in hospital stay was associated with a 4% increase (AOR: 1.04; 95% CI 1.01–1.08; $P = 0.024$). Overall, this model was found to have good discrimination with an area under the receiver operating characteristic of 0.83.

4. Discussion

Although it has been previously supported that patients presenting with cervical spinal cord injuries are at increased risk for dysphagia development [1–9], no investigations, to the authors' knowledge, have examined the implications of non-spinal cord–affected traumatic CSI (including vertebral fracture and ligamentous injury) on incidence of this complication. This study sought to add to the literature on this underrepresented facet by evaluating clinically diagnosed dysphagia trends within a total CSI population, considering the impact of demographic factors including spinal cord injury status and age on dysphagia development. Although our adjusted analysis supports the existing literature that posits injury to the cervical spinal cord increases a patient's risk for dysphagia development [1–9], only 19% of our dysphagic subpopulation had cord injuries. The vast majority of this subgroup (81%) developed dysphagia without any evidence of cervical spinal cord injury. This finding supports our hypothesis that although a greater rate of swallowing dysfunction would be observed in the spinal cord–injured CSI subgroup (23/56; 41%), dysphagia would still be a major complication for CSI patients presenting without damage to the spinal cord (100/425; 24%). This large percentage of non-spinal cord–injured dysphagic patients is a significant finding considering the literature that suggests rates of dysphagia ranging from 17% to 41% within the spinal cord–injured specific population [1–4]. When considering the impact of age on dysphagia development, we found significantly higher rates of clinically diagnosed swallowing dysfunction within the geriatric (90/227; 40%) compared with the general (33/254; 13%) population—with the geriatric population comprising 73% (90/123) of our dysphagic subgroup. This finding also supports our hypothesis, and a growing body of literature [1,3], suggesting geriatric traumatic CSI patients are at heightened risk for dysphagia development compared with the general population (Fig.).

Although the literature is replete with investigations analyzing the prevalence of dysphagia within cervical spinal cord–injured and age-defined subgroups, differences in identified rates and trends among traumatically injured populations are common [1–4]. In a 1999 study conducted by Kirshblum *et al.* [1] examining rates of swallowing deficits within a 187 sample cervical spinal cord–injured population, 17% of patients were found to exhibit dysphagia. In 2003, Wolf and Meiners [4] analyzed a smaller cohort of cervical spinal cord–injured patients ($n = 51$) and found a much higher incidence of dysphagia development at 41%. Similarly, in 2004,

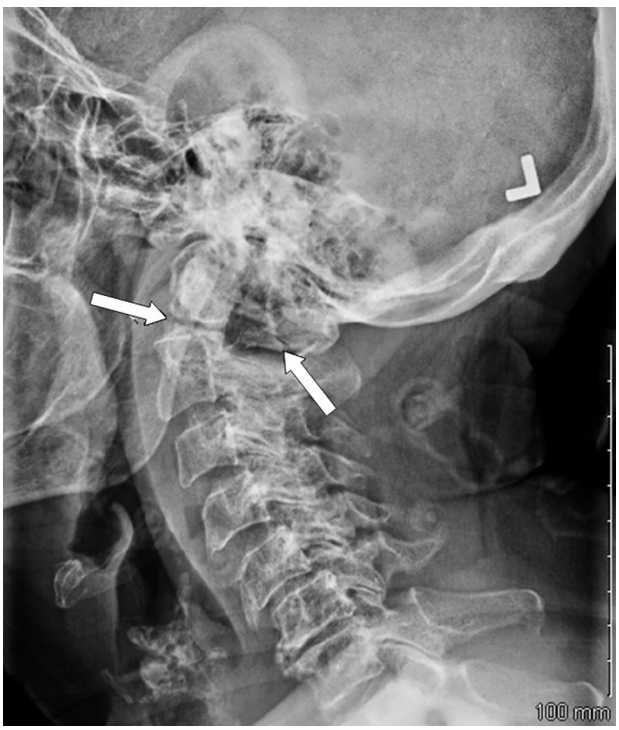

Fig. – Geriatric traumatic CSI study population patient with C1 and C2 fractures (arrow pointers) who developed dysphagia without evidence of spinal cord injury.

Abel *et al.* [2] examined a sample of 75 patients with cervical spinal cord injuries and found a 35% prevalence of dysphagia development. More recently, Shin *et al.* [3] in 2011, found a 29% dysphagia rate within a sample of 121 spinal cord–injured patients. Considering only the spinal cord–injured subgroup analyzed in our investigation ($n = 56$), our dysphagia rate of 41% is on the upper end of the literature detailed previously [2,4]. Even when broadening the analysis to our entire study population (including nonspinal cord–injured CSI patients), the overall dysphagia rate of 26% still falls within the range of the existing literature analyzing only spinal cord–injured populations [1–4]. This suggests that although dysphagia is a more common complication after spinal cord injury, it is likely to have some implications within nonspinal cord–injured CSI populations as well. Had the aforementioned investigations analyzed all CSI patients, we would be able to directly compare our findings, but as mentioned previously, we were unable to locate any existing literature analyzing such a population.

In addition to the dysphagia rate discrepancies found within the literature for traumatically injured populations, conflicting evidence regarding the impact of age on dysphagia development in these analyses is prevalent. Although it is generally accepted that age is significantly associated with atraumatic development of dysphagia as a result of the physiological changes that naturally occur throughout aging process [21–26], in review of the previously discussed literature, only Kirshblum *et al.* [1] were able to definitively correlate age and risk of dysphagia development within a traumatically injured spinal cord population ($P = 0.028$). Wolf and Meiners [4] and Abel *et al.* [2] reported no association between age and dysphagia. Shin *et al.* [3] noted the trend and reasonability of age as a risk factor for dysphagia development but was unable to show statistical significance beyond an increased risk for geriatric patients to aspirate once developing dysphagia. Our results support those of Kirshblum *et al.* [1], and the conjecture of Shin *et al.* [3] as age was found to be a significant predictor of dysphagia development in both univariate (OR: 1.03, 95% CI 1.02–1.04; $P < 0.001$) and multivariate (AOR: 1.06, 95% CI 1.04–1.07; $P < 0.001$) analyses. Along with age, injury to the spinal cord, ISS, GCS, TBI, facial fracture, ventilator use, hospital LOS, halo vest fixation, and tracheostomy were all found to be significant predictors of dysphagia in univariate analysis, as could be expected based on existing literature [1–20]. Past medical history of stroke, although strongly supported in the literature as a predictor for atraumatic dysphagia development [27–29], was found nonsignificant within this traumatically injured population. It is possible that our small sample of only four patients who previously suffered a stroke confounded these results, however. On inputting the aforementioned significant dysphagia predictors into a multivariate logistic regression model, some unexpected results were found. Whereas age, GCS, TBI, facial fracture, spinal cord injury, hospital stay, ventilator use, and halo vest fixation remained significant predictors of dysphagia, ISS and tracheostomy fell out of statistical significance. Considering tracheostomy is a relatively well-supported predictor of dysphagia within traumatically injured populations [1,3,15], we were surprised to see this variable fall from significance while controlling for other demographic and injury severity covariates. As with stroke history, it is possible that our small sample of patients with tracheostomy, as well as our practice protocol of only testing patients for dysphagia after tracheostomy removal, contributed to this result.

As with any retrospective analysis, this investigation has its inherent limitations. In addition to our small, single institution–derived sample, Lancaster, Pennsylvania is a relatively homogenous, geriatric haven. As such, the mean age of CSI patients within our study population is relatively old—more so than previous studies analyzing dysphagic trends within cervical spinal cord–injured specific populations. Although multivariate regression models were used to control for demographic factors within our analysis, it is possible that our substantial geriatric population could have impacted dysphagic trends identified within our results. In addition, due to the fact that there is currently no formalized dysphagia screening protocol for patients presenting with CSIs within our institution, it is likely that the estimated dysphagia prevalence reported within this investigation is conservative. It is important to note that we have not reported the true incidence of dysphagia within our hospital, rather the rate of clinically diagnosed dysphagia.

5. Conclusion

The association between cervical spinal cord injury and dysphagia development is a well-documented clinical reality [1–9]. Although it is generally accepted that dysphagia screening should be conducted in patients presenting with cervical spinal cord injuries, our results suggest that all CSI patients, especially those aged older than 65 y, should be placed at heightened suspicion. Within our total CSI population, we identified clinically diagnosed rates of dysphagia that were consistent with or exceeding previously reported rates within strictly spinal cord–injured populations. Only considering the presence of dysphagia for those with spinal cord injuries may neglect a large percentage of the overall dysphagic population, placing patients at increased risk for developing a host of secondary complications.

Authors' contributions: J.C.L. contributed to study design, data interpretation, and article preparation. B.W.G. contributed to study design, data collection, data analysis, data interpretation, and article preparation. K.J.R. contributed to study design, data collection, data interpretation, and article preparation. A.R.V., A.V., J.A., and M.G. contributed to data collection, data interpretation, and article preparation. F.B.R. contributed to study design, data interpretation, article preparation, and editorial oversight.

Disclosure

The authors report no proprietary or commercial interest in any product mentioned or concept discussed in this article.

REFERENCES

[1] Kirshblum S, Johnston MV, Brown J, O'Connor KC, Jarosz P. Predictors of dysphagia after spinal cord injury. Arch Phys Med Rehabil; 1999.
[2] Abel R, Ruf S, Spahn B. Cervical spinal cord injury and deglutition disorders. Dysphagia 2004;19:87.
[3] Shin JC, Yoo JH, Goo HR, Kim DH. Dysphagia in cervical spinal cord injury. Spinal Cord 2011;49:1008.
[4] Wolf C, Meiners TH. Dysphagia in patients with acute cervical spinal cord injury. Spinal Cord 2003;41:347.
[5] Gragic V. Cervicogenic dysphagia: swallowing difficulties caused by functional and organic disorders of the cervical spine. Liejec Vjesn 2013;135:92 [Article originally in Croatian].
[6] Seidel RO, Nusser-Muller-Busch R, Kurzweil M, Niedeggen A. Dysphagia in acute tetraplegics: a retrospective. Spinal Cord; 2009:197.
[7] Shem K, Castillo K, Wong SL, Chang J, Kolakowsky-Hayner S. Dysphagia and respiratory care in individuals with tetraplegia: incidence, associated factors, and preventable complications. Top Spinal Cord Inj Rehabil 2012;18:15.
[8] Papadopoulou S, Exarchakos G, Beris A, Ploumis A. Dysphagia associated with cervical spine and postural disorders. Dysphagia 2013;28:469.
[9] Shem K, Castillo K, Wong S, Chang J. Dysphagia in individuals with tetraplegia: incidence and risk factors. J Spinal Cord Med 2011;34:85.
[10] Garon BR, Sierzant T, Ormiston C. Silent aspiration: results of 2000 video fluoroscopic evaluations. J Neurosci Nurs 2009;41:178.
[11] Altman KW, Yu GP, Schaefer SD. Consequence of dysphagia in the hospitalized patient: impact on prognosis and hospital resources. Arch Otolaryngol Head Neck Surg 2010;136:784.
[12] Rofes L, Arreola V, Almirall J, et al. Diagnosis and management of oropharyngeal dysphagia and its nutritional and respiratory complications in the elderly. Gastroenterol Res Pract; 2011:1.
[13] Cakir E, Hatipoglu ON, Yilman I, Eker A, Tansel O, Sut N. Hospital-acquired pneumonia developed in non-intensive care units. Respiration 2009;78:416.
[14] Marik PE, Kaplan D. Aspiration pneumonia and dysphagia in the elderly. CHEST 2011;124:328.
[15] Chaw E, Shem K, Castillo K, Wong S, Chang J. Dysphagia and associated respiratory considerations in cervical spinal cord injury. Top Spinal Cord Inj Rehabil 2012;18:291.
[16] Higgins DM, Maclean JC. Dysphagia in patients with a tracheostomy: six cases of inappropriate cuff deflation or removal. Heart Lung 1997;26:215.
[17] Bonanno PC. Swallowing dysfunction after tracheostomy. Ann Surg 1971;174:29.
[18] Morishoma N, Ohota K, Miura Y. The influences of halo-vest fixation and cervical hyperextension on swallowing in healthy volunteers. Spine 2005;30:179.
[19] Stambolis V, Brady S, Klos D, Wesling M, Fatianov T, Hildner C. The effects of cervical bracing upon swallowing in young, normal, healthy volunteers. Dysphagia 2003;18:39.
[20] Bradley JF, Jones MA, Farmer EA, et al. Swallowing dysfunction in trauma patients with cervical spine fractures treated with halo-vest fixation. J Trauma 2011;70:46.
[21] Lingren S, Janzon L. Prevalence of swallowing complaints and clinical findings among 50-79-year-old men and women in an urban population. Dysphagia 1991;6:187.
[22] Talley NJ, Weaver AL, Zinsmeister AR, Melton LJ. Onset and disappearance of gastrointestinal symptoms and functional gastrointestinal disorders. Am J Epidemiol 1992;136:165.
[23] Tibbling L, Gustafsson B. Dysphagia and its consequences in the elderly. Dysphagia 1991;6:200.
[24] Achem SR, Devault KR. Dysphagia in aging. J Clin Gastroenterol 2005;39:357.
[25] Logemann JA, Curro FA, Pauloski B, Gensler G. Aging effects on oropharyngeal swallow and the role of dental care in oropharyngeal dysphagia. Oral Dis 2013;19:733.
[26] Jahnke V. Dysphagia in the elderly. HNO 1991;39:442.
[27] Martino R, Foley N, Bhogal S. Dysphagia after stroke: incidence, diagnosis, and pulmonary complications. Stroke 2005;36:2756.
[28] Mann G, Hankey GJ, Cameron D. Swallowing function after stroke: prognosis and prognostic factors at 6 months. Stroke 1999;30:744.
[29] Leder SB, Espinosa JF. Aspiration risk after acute stroke: comparison of clinical examination and fiberoptic endoscopic evaluation of swallowing. Dysphagia 2002;17:214.