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ORIGINAL COMMUNICATION

Dysphagia in patients with acute striatocapsular hemorrhage

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Abstract Dysphagia is found in up to 80% of acute stroke patients. To date most studies have focused on ischemic stroke only. Little is known about the incidence and pattern of dysphagia in hemorrhagic stroke. Here we describe the characteristics of dysphagia in patients with striatocapsular hemorrhage. Fiberoptic Endoscopic Evaluation of Swallowing (FEES) was carried out in 30 patients with acute striatocapsular hemorrhage. Dysphagia was classified according to the six-point Fiberoptic Endoscopic Dysphagia Severity Scale (FEDSS) within 72 h after admission. Lesion volume, hemisphere and occurrence of ventricular rupture were determined from computer tomography scans. Data on initial NIH-SS, clinical symptoms, need for endotracheal intubation, diagnosis of pneumonia and feeding status on discharge were recorded. Swallowing impairment was observed in 76.7% of patients ($n = 23$). Mean FEDSS score was 3.1 ± 1.5. Main findings were penetration or aspiration of liquids as well as leakage to valleculae and piriform sinus. Incidence of pneumonia was 30.0% ($n = 9$). Age, NIH-SS and hematoma volume did not correlate with dysphagia severity. None of the clinical characteristics was predictive for dysphagia. On discharge after 12.9 ± 5.3 days, a two-point improvement on the FEDSS was seen in seven patients, (30.4%) and five patients (21.7%) had gained at least one point. In striatocapsular hemorrhage, dysphagia is a common and so far underrecognized symptom. FEES results indicate predominant impairment of oral motor control. Swallowing impairment is not related to other clinical deficits, stroke severity or lesion characteristics. Thus, detailed dysphagia assessment is indicated in all cases.

Keywords Dysphagia · Stroke · Intracerebral hemorrhage · Swallowing assessment · FEES

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Introduction

Dysphagia is an important symptom of acute stroke, with a reported incidence ranging between 29 [5] and 81% [44] depending on the diagnostic criteria, timing and method of assessment, and stroke features in the respective patient population [43]. Due to malnutrition [11, 12] and aspiration, disturbed swallowing is associated with the development of chest infection, prolonged hospital stay, increased mortality and poor long-term outcome (i.e., dependency and nursing home admission) [30, 40, 43, 52, 53]. It has been shown that early detection of swallowing impairment guiding decisions about feeding strategy and leading towards timely treatment is associated with a more favorable outcome in dysphagic stroke patients [8, 18, 22, 29]. Therefore, current research focuses on investigating the relationship between stroke characteristics and dysphagia features in order to identify predictors for impaired swallowing and to distinguish those patients who have a special need for further evaluation or intervention. However, presumably due to its higher incidence, many studies have been focused on ischemic strokes only [39, 54]. Others included a small number of hemorrhagic stroke cases, but did not analyze them as a separate

group regarding swallowing disturbance patterns [28, 40, 41, 49]. There is only one study, by Smithard et al. (2007) [53], that shows that patients classified as "not being safe to swallow" were more likely to have had an intracranial hemorrhage of any location including subarachnoid hemorrhage than an ischemic infarction.

The striatocapsular area is of particular interest as it is the most frequently affected site of spontaneous intracerebral hemorrhage caused by the main risk factor, hypertension [4, 9, 17, 50]. Although this area is supplied by a variety of arteries, it is still referred to as a single clinical entity and also known as basal ganglionic hemorrhage [9].

Thus, the purpose of this study was to describe the characteristics of dysphagia in patients with intracerebral hemorrhage of the striatocapsular area using Fiberoptic Endoscopic Evaluation of Swallowing (FEES), which has proved to be an excellent bedside method for a quick, safe and precise dysphagia assessment in the first days after stroke [16, 36, 38, 59–61].

Patients and methods

Subjects

Thirty consecutive first-ever stroke patients meeting the inclusion criteria were enrolled in this study. Patients were eligible to be included if they had an isolated striatocapsular hemorrhage diagnosed by CT scan on admission. The striatocapsular area was defined as an area including the caudate nucleus, putamen, globus pallidus, anterior and posterior limbs of the internal capsule and subinsular area [9, 24]. Lesion side and the presence of ventricular rupture were determined. Hematoma size was calculated using the ABC/2 method where A is the greatest hemorrhage diameter by CT, B is the diameter 90° to A, and C is the approximate number of CT slices with hemorrhage multiplied by the slice thickness [23, 33]. Exclusion criteria were symptom onset more than 24 h before admittance to our stroke unit, preexisting swallowing dysfunction, a severely reduced state of consciousness and endotracheal intubation on admission. Stroke severity was measured on admission using the National Institutes of Health Stroke Scale (NIH-SS) [7].

All patients were evaluated by FEES within 72 h after admission. Stroke symptoms, later need for endotracheal intubation, occurrence of pneumonia and feeding status by the time of discharge from our hospital were documented. The diagnosis of pneumonia was based on the presence of three or more of the following features during hospital stay: fever (>38°C), productive cough with purulent sputum, abnormal respiratory examination [tachypnea (>22/min), tachycardia, inspiratory crackles, bronchial breathing], abnormal chest radiograph, arterial hypoxemia ($PO2 < 9.3$ kPa), or isolation of a relevant pathogen and use of antibiotics [40]. Informed consent was obtained from all subjects, or their next of kin, in case the patient's communication was impaired.

Equipment

Equipment consisted of a 3.1-mm-diameter flexible fiberoptic rhinolaryngoscope (ENF-P4, Olympus, Hamburg, Germany), light source (Endovision Telecam, SL PAL 20212020, Storz, Tuttlingen, Germany), camera (Endovision Telecam, SL PAL 20212030, Storz), color monitor (DVM 14M2MDE, Sony, Tokyo, Japan) and video recorder (SVO9500MDP, Sony). All examinations were recorded and stored on hard disc for later review.

Dysphagia assessment

All subjects were endoscopically assessed by a trained neurologist together with a speech language pathologist in accordance with our protocol for fiberoptic endoscopic assessment of dysphagia in acute stroke patients, which we had previously developed and evaluated [16, 59, 60]. Following this protocol each examined patient was classified according to our six-point fiberoptic endoscopic dysphagia severity scale (FEDSS) that allows a quick deduction of clinical consequences. The detailed protocol is described elsewhere [16, 59]. In brief, the examination starts with rating the severity of oropharyngeal secretions. In case saliva pooling with penetration or aspiration was found, severe dysphagia was suspected, and a score of 6 was given. Patients who were able to handle their saliva without penetration or aspiration received a teaspoon of puree consistency next. Those who showed penetration or aspiration without sufficient protective reflex (i.e., coughing or swallowing) on at least one of three attempts were again diagnosed with severe dysphagia (score 5). If sufficient protective reflexes were present, score 4 was attributed. Patients managing puree consistency without any aspiration events were exposed to a teaspoon of colored water. Its penetration or aspiration without sufficient protective reflex kept the patient on his former score, while the presence of protective reflexes led to score 3. If patients were able to swallow liquids three times without penetration or aspiration, a small piece of white bread was given to them at the last step. Here, penetration or aspiration or massive residues (>50% of bolus size) in the valleculae or piriform sinus were taken as evidence of severe difficulty with this food consistency, resulting in score 2. If none of these findings were observed on three consecutive trials, a score

of 1 was given. Any score above 1 on the FEDSS was classified as dysphagia.

Each of the six scores is accompanied by specific recommendations concerning protective and rehabilitative measures. For example, scores 4–6 are all indicative of severe dysphagia that forbids an oral diet (score 6: no oral food; endotracheal intubation maybe necessary in case of respiratory distress; score 5: nil by mouth; score 4: small amount of pureed food during swallowing therapy only). On the other hand, scores 1–3 allow for an early oral feeding with a specifically adapted diet (score 3: puree consistency only, score 2: soft solid food, score 1: normal diet) [16].

According to the FEES standard protocol proposed by Langmore [35, 37], salient parameters of swallowing function such as premature spillage and delayed swallowing reflex were also examined. As described in detail elsewhere [35, 58], the swallowing reflex was rated as delayed if both the norms of spillage frequency to the pyriforms and the normal average dwell time in the pyriforms (i.e., 1.5 s) were exceeded. A disturbed oral bolus control was determined when food and/or liquid boluses spilled over the base of tongue into the hypopharynx during the oral swallowing stage, long before the swallow was initiated (test of oral containment).

Statistical analysis

Descriptive statistics were used to quantify patient characteristics and salient videoendoscopic findings. Binary logistic regression analysis was applied to test for significant associations among stroke symptoms, presence of ventricular rupture and lesion side with occurrence of penetration/aspiration and dysphagia in general. Spearman correlation analyses were performed to investigate the relation among age, NIH-SS, hematoma volume and FEDSS. The analyses were carried out using PASW Statistics 18.0.

Results

Demographic and clinical features as well as stroke characteristics of the 30 patients enrolled in the study are presented in Table 1. The average NIH-SS of our study population was 11 points, indicating a prominent neurological deficit in most of the included patients. The mean hematoma volume was 14.71 ml, and ventricular rupture occurred in five cases. One patient was managed surgically (ventricular drain). Four patients required endotracheal intubation after FEES because of insufficient protective reflexes ($n = 1$), respiratory failure ($n = 2$) and need for neurosurgical intervention ($n = 1$). Pneumonia was diagnosed in nine cases (30.0%).

The period of time from admission to FEES performance was less than 48 h on average. The procedure was tolerated well by all patients. Frequencies of salient videoendoscopic findings and the resulting FEDSS score are displayed in Table 2. The overall incidence of dysphagia (i.e., FEDSS > 1) was 76.7% ($n = 23$). The mean FEDSS score was 3.1 ± 1.5. Except for one patient who showed pooling of secretions and silent aspiration of saliva, at least one food consistency could be tested in all subjects. One third of the patients showed penetration of puree consistency, while aspiration without sufficient protective reflex only happened in one case so that the examination was stopped at this point. With liquids, penetration occurred in almost two-thirds of the 28 remaining patients, and 39.3% of these showed aspiration as well. The majority of subjects ($n = 23$, 76.7%) were found with leakage to the valleculae, even reaching into the pirifom sinus in 16 cases. In two patients, isolated leakage to the valleculae was observed, which does not count for the FEDSS score. Thus, a score of 1 was given. Penetration and severe residue were uncommon in the ten patients receiving soft solid food, and aspiration was not noticed. According to their respective FEDSS score, 14 patients were put on "nil by mouth" and enteral tube feeding was started; 8 of these patients had small amounts of pureed food during swallowing therapy only. Six subjects were able to manage pureed food, and in three patients soft solid food could be given. Seven subjects demonstrated no relevant signs of dysphagia, and dietary adaptations were not necessary.

Presence of ventricular rupture, lesion side and stroke symptoms did not predict dysphagia in general or the occurrence of penetration/aspiration in particular. Patients' age inversely correlated with hematoma volume ($p < 0.05$) and NIH-SS on admission ($p < 0.01$), whereas NIH-SS and hematoma volume showed a strong positive correlation ($p < 0.01$) (see Table 3). None of these three parameters significantly related to FEDSS score.

By the time of discharge from our hospital, four patients (13.3%) were nourished via a percutaneous endoscopic gastrostomy (PEG) tube, and two patients (6.7%) were still on nasogastric tube feeding. Pureed food was managed by eight subjects (26.7%), and six subjects (20.0%) were on soft solid food. Ten patients (33.3%) were on a normal diet. Regarding the 23 patients with dysphagia on admission, 30.4% ($n = 7$) gained two points on the FEDSS, and 21.7% ($n = 5$) improved at least for one point. In 43.5% ($n = 10$), dysphagia severity remained unchanged, and a single subject changed one point for the worse. Mean duration of

Table 1 Main characteristics of study population

Patient characteristics	
Total, *n* (%)	30 (100.0)
Sex: male/female, *n* (%)	16/14 (53.3/46.7)
Age (years), mean ± SD	71.13 ± 10.05
NIH-SS (points), mean ± SD	11.33 ± 4.51
Hematoma volume (ml), mean ± SD	14.741 ± 11.506
Lesion side: left/right, *n* (%)	18/12 (60.0/40.0)
Ventricular rupture: yes/no, *n* (%)	5/25 (16.7/83.3)
Etiology	
Hypertension, *n* (%)	25 (83.3)
Anticoagulant therapy, *n* (%)	5 (16.7)
Symptoms	
Hemiparesis, *n* (%)	29 (96.7)
Facial palsy, *n* (%)	29 (96.7)
Aphasia, *n* (%)	11 (36.9)
Dysarthria, *n* (%)	21 (70.0)
Neglect, *n* (%)	9 (30.0)
Time to FEES	
≤24 h, *n* (%)	14 (46.7)
>24–≤48 h, *n* (%)	11 (36.7)
>48–≤72 h, *n* (%)	5 (16.7)

hospitalization was 12.9 ± 5.3 days. There were no in-hospital deaths.

Discussion

Our results show that clinically significant dysphagia is a common symptom in striatocapsular hemorrhage with an incidence that is among the highest reported so far for a specific lesion location [41, 43]. Only in brainstem stroke was dysphagia found in more than 80% of cases [44]. Subjects had predominant impairment of the oral swallowing phase with premature leakage as a main finding. The mean FEDSS score of our study population was within the range of the results of former studies (2.29 ± 1.57 [59] to 3.96 ± 1.40 [16]) in which a mixed stroke patient population (i.e., hemorrhagic and ischemic) had been involved. This is striking as the basal ganglia and internal capsule are not brain areas typically linked to swallowing at first sight and up to now dysphagia was not recognized as a typical symptom in striatocapsular hemorrhage [51]. Several studies using different swallowing tasks and imaging methods demonstrated activation in this area during deglutition [26, 46, 47, 55, 63], whereas others did not [27, 42]. Clinical studies show increasing evidence for the relevance of subcortical lesions as the cause of dysphagia [10, 14, 39, 48, 62]. The basal ganglia functionally

Table 2 Frequencies of salient videoendoscopic findings and resulting FEDSS score

	n (% of all subjects)
Handling of secretions	30[a] (100.0)
Saliva pooling	1 (3.3)
Aspiration of saliva	1 (3.3)
Puree consistency	29[a] (96.7)
Penetration	11 (36.7)
Aspiration	1 (3.3)
Liquids	28[a] (93.3)
Penetration	18 (60.0)
Aspiration	11 (36.7)
Soft solid food	10[a] (33.3)
Penetration	1 (3.3)
Aspiration	0 (0.0)
Leakage	28[a] (93.3)
To valleculae	23 (76.7)
To piriform sinus	16 (53.3)
Without delayed swallowing reflex	10 (33.3)
With delayed swallowing reflex	6 (20.0)
Residue	10[a] (33.3)
In valleculae	2 (6.7)
In piriform sinus	0 (0.0)
Delayed swallowing reflex (total)	9 (30.0)
Cough reflex	30[a] (100.0)
Insufficient	5 (16.7)
Missing	3 (10.0)
FEDSS score	
1	7 (23.3)
2	3 (10.0)
3	6 (20.0)
4	8 (26.7)
5	5 (16.7)
6	1 (3.3)

[a] Number of patients exposed

connect the cerebral cortex and the thalamus with one likely function being gating of sensory input to achieve motor control in deglutition [31, 55]. It seems that damage of the basal ganglia and internal capsule in our patients mainly led to disturbed oral motor control causing premature leakage in the majority of subjects. In accordance with our results Steinhagen et al. (2009) [54] comparing clinical and videoendoscopic dysphagia patterns with predefined lesion locations found that infarctions of basal ganglia and the internal capsule were associated with buccofacial apraxia, also resulting in oral phase dysphagia. Han et al. (2005) [28] reported that patients with recurrent cortico-subcortical stroke exhibited a significantly higher rate of abnormalities in the oral phase, including leakage,

Table 3 Results of Spearman correlation analysis

	NIH-SS rho (p value)	Hematoma volume rho (p value)	FEDSS rho (p value)
Age	−0.489 (0.006)†	−0.399 (0.029)*	0.142 (0.455)
NIH-SS		0.506 (0.004)†	−0.096 (0.612)
Hematoma volume			0.046 (0.811)

* Significant ($p < 0.05$), two-tailed

† Significant ($p < 0.01$), two-tailed

than their counterparts with brainstem stroke, which is known to cause mainly pharyngeal phase dysphagia [3, 34, 56]. Moreover, Cola et al. (2010) [10] stated that swallowing deficits involving oral control and transfer might be a marker of subcortical neural axis involvement. Thus, there is growing evidence that impairment of oral motor control is a characteristic symptom of striatocapsular lesions.

Presence of dysphagia in general as well as penetration and aspiration in particular were not predicted by other clinical symptoms. This may partly be due to the fact that the small patient population was quite homogenous regarding their clinical deficits, and some symptoms like facial palsy were present in almost every subject. Pneumonia incidence of 30% in our study population was within the upper range of previously reported findings in acute stroke [15, 28, 52, 59]. More than 43% of patients aspirated. In support of our results other authors found that subcortical and especially basal ganglionic infarctions are significantly associated with occurrence of aspiration [1, 14] and poststroke infection [57]. Moreover, Minnerup et al. (2010) [45] reported a positive correlation between intracerebral hemorrhage and incidence of pneumonia and infections in general.

There is an ongoing debate whether dysphagia severity and deficits in a particular stage of swallowing are related to lesion side. Concerning subcortical stroke, prior research has suggested left hemispheric infarction to be associated with a higher degree of dysphagia [10], although the sample size in this study was small, and the power is therefore limited. We did not find any association between lesion side and dysphagia. Ventricular rupture and lesion volume are generally associated with a worse outcome [6, 13, 17]. However, in our study these factors were not related to the FEDSS score. This may be due to the small sample size and a selection bias: subjects with large hematoma and extensive ventricular rupture were more likely to be intubated on admission and therefore excluded from the trial. The same is possibly true for the reciprocal association between age and NIH-SS or hematoma volume. The older the patients, the more likely they were to be excluded because of a severely reduced state of consciousness [2]. In a recent study by Kiphuth et al. [32], neurocritical care patients with spontaneous intracerebral hemorrhage were investigated searching for predictors of later percutaneous endoscopic gastrostomy (PEG) tube placement. Their results partly go along with ours because besides lobar hemorrhages with increasing volume, there was no other hematoma site, and especially not hematoma volume, which was associated with PEG tube placement.

So far we have little information on the natural history of recovery of swallowing deficits after purely subcortical or hemorrhagic stroke. We show for the first time data of the clinical course of dysphagia and its resulting nutritional adaptations in the acute phase after striatocapsular hemorrhage. Cola (2010) [10] observed that, despite receiving swallowing treatment, all of their four dysphagic patients suffering from subcortical infarction remained on an unaltered diet throughout hospitalization. Others hypothesized that dysphagia resulting from acute disconnection between the cortex and brain stem is likely to improve quickly as compared to dysphagia resulting from damage to important primary areas for swallowing control [25]. This seems to be true for our study population: within 2 weeks more than half of our initially dysphagic patients ($n = 23$) gained at least one point on the FEDSS, indicating substantial short-term recovery. By the time of discharge 74% were on an oral diet; only 26% were still on tube feeding. These results are in line with another bigger study tracking clinical improvement in a mixed stroke population (i.e., ischemic and hemorrhagic, cortical and subcortical) [21]. However, it is still unknown whether ischemia and hemorrhage disturb swallowing to the same extent. Overall comparison of both types of stroke showed no differences with respect to incidence of dysphagia [19] and outcome parameters like malnutrition [20] or survival [30]. But to address this matter suitably it would be essential to compare dysphagia characteristics in both conditions with matched lesion location.

Conclusion

Dysphagia mainly due to impaired oral motor control is a common and so far underrecognized symptom in striatocapsular hemorrhage. Infectious complications frequently arise. Since clinical parameters were not related to the incidence and severity of swallowing dysfunction, every patient must be considered at risk for dysphagia. We therefore recommend that all patients with striatocapsular

hemorrhage should receive detailed swallowing assessment within the first 3 days after symptom onset. There is evidence from our data that dysphagia in this patient group has a substantial potential for short-term improvement. Whether the type of stroke, lesion location or size is related to specific videoendoscopic dysphagia characteristics is still debated. Further studies in patients with a predefined type of intracerebral hemorrhage (e.g., lobar, pontine) are needed to clarify this issue.

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

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