Original Contribution

Stereotactic Catheter Ventriculocisternostomy for Clearance of Subarachnoid Hemorrhage

A Matched Cohort Study

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Background and Purpose—Delayed cerebral infarction (DCI) is a major source of morbidity and mortality after aneurysmal subarachnoid hemorrhage. We report a novel intervention—stereotactic catheter ventriculocisternostomy (STX-VCS) and fibrinolytic/spasmolytic lavage therapy—for DCI prevention. Outcomes of 20 consecutive patients are compared with 60 matched controls.

Methods—On the basis of individual treatment decisions, STX-VCS was performed in 20 high-risk aneurysmal subarachnoid hemorrhage patients admitted to our department between September 2015 and October 2016. Three controls matched for age, sex, aneurysm treatment method, and admission Hunt and Hess grade were assigned to each case treated by STX-VCS. DCI was the primary outcome. Mortality and mRS at rehabilitation discharge were secondary outcome parameters. The association between STX-VCS and DCI, mortality, and mRS was assessed by conditional logistic regression.

Results—Stereotactic procedures were performed without surgical complications. Continuous cisternal lavage was feasible in 17 of 20 patients (85%). One adverse event because of cisternal lavage was without sequelae. DCI occurred in 25 of 60 (42%) controls and 3 of 20 (15%) patients with STX-VCS (odds ratio, 0.15; 95% confidence interval, 0.04–0.64). Mortality occurred in 20 of 60 (33%) controls and 1 of 20 (5%) patients with STX-VCS, respectively (odds ratio, 0.08; 95% confidence interval, 0.01 – 0.66). Favorable outcome (mRS≤3) at rehabilitation discharge was observed in 12 of 20 patients with STX-VCS (60%) versus 21 of 60 (35%) matched controls (odds ratio, 0.26; 95% confidence interval, 0.8–0.86).

Conclusions—STX-VCS was feasible and safe in patients with severe aneurysmal subarachnoid hemorrhage. Initial results indicate that DCI and mortality can be reduced, and neurological outcome may be improved with this method. **(*Stroke*. 2017;48:00-00. DOI: 10.1161/STROKEAHA.117.018397.)**

Key Words: aneurysm ■ confidence intervals ■ odds ratio ■ patient discharge ■ subarachnoid hemorrhage ■ vasospasm

Aneurysmal subarachnoid hemorrhage (aSAH) is a hemorrhagic stroke associated with high morbidity and mortality.[1] Delayed cerebral infarction (DCI) is an important cause of morbidity and mortality in survivors of the initial bleeding event.[2] Currently available treatments for DCI prevention are insufficient.[2]

DCI results from cerebral vasospasm putatively triggered by blood break-down products in the subarachnoid space. Therefore, treatments directed at the elimination of subarachnoid blood might represent promising approaches.[3] Surgical access to the brain for aneurysm clipping allows for placement of a cisternal catheter and administration of blood-clearing therapies.[3] In contrast, a safe access to the basal cisterns of the brain after aneurysm coiling does not exist.[4] Today, most aneurysms are secured by coiling. Therefore, the majority of patients with aSAH are currently not eligible for cisternal treatments.

To create a continuous treatment access to the subarachnoid space—irrespective of aneurysm location and treatment method—we performed stereotactic catheter ventriculocisternostomy (STX-VCS) and lavage therapy in 20 patients with aSAH at high risk for DCI.

Patients and Methods

The Institutional Review Board of our University Medical Center was consulted before the first implementation of STX-VCS in patients with aSAH. The experimental therapy was

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hereafter offered to high-risk aSAH patients on the basis of individual treatment decisions. Institutional review board approval for retrospective analyses was obtained.

The study is reported according to the STROBE guidelines (Strengthening the Reporting of Observational Studies in Epidemiology) for reporting of observational studies.[5] The experimental therapy consisting of STX-VCS and lavage therapy was introduced for the treatment of patients with aSAH in our department in September 2015[6]. Based on case-by-case risk evaluations, this novel therapy was offered to patients at high risk for DCI and mortality (eg, Hunt and Hess grade, ≥3; modified Fisher grade, ≥3) and an external ventricular drain in situ. Only patients with successful aneurysm securing by endovascular coiling or microsurgical clipping were included. We did not offer the experimental therapy to patients with a per se dismal prognosis (eg, postictal cardiopulmonary resuscitation or signs of brain stem herniation for >60 minutes and massive intracerebral hemorrhage). The legal representatives of all patients enrolled in the individual treatment decisions provided informed consent. The 20 patients with STX-VCS reported in this study were selected from 58 consecutive patients with (1) subarachnoid hemorrhage of aneurysmal origin, (2) admitted within 72 hours of aSAH onset, and (3) wish for treatment, who were admitted to our department between September 2015 and October 2016 (Figure 1).

The experimental therapy was performed as previously described[6]: fibrinolytic lavage using electrolyte solution (Jonosteril; Fresenius-Kabi GmbH, Bad Homburg, Germany) containing 100 IU/mL Urokinase was administered at a rate of 50 to 100 mL/h, typically for 14 days. Irrigation without Urokinase was continued until removal of the catheter, usually after 21 days. In case of increased mean flow velocity (MFV) of either proximal cerebral artery ≥160 cm/s in transcranial doppler, spasmolytic lavage therapy containing nimodipine (0.005 mg/mL; Bayer Vital GmbH, Leverkusen, Germany) was administered. Intensive care management was conducted according to current guidelines.[7]

Presence or absence of DCI was assessed by an independent neurologist (W.-D.N.) and neuroradiologist (K.E.) who was not involved in the management of the patients and blinded to the treatment. DCI rating was performed according to the Vergouwen criteria. DCI was classed as the presence of cerebral infarction on computed tomography (CT) or magnetic resonance imaging within 6 weeks after aSAH, or on the latest CT or magnetic resonance imaging before death within 6 weeks, not present on the CT or magnetic resonance imaging scan between 24 and 48 hours after early aneurysm occlusion and not attributable to other causes, such as surgical clipping or endovascular treatment. Hypodensities on CT imaging resulting from ventricular catheter or intraparenchymal hematoma are not regarded as DCI.[8] If no imaging 24 to 48 hours after early aneurysm occlusion was available, an expert opinion was made by the independent raters.

Neurological outcome was evaluated by mRS. mRS rating used our discharge reports for patients with in-hospital mortality. Medical discharge reports from rehabilitation facilities were retrieved for all surviving patients included in the present study. All documents were anonymized, and all calendaric information and referencing about the experimental therapy were concealed. The mRS was determined by a certified mRS rater (W.-D.N.). Median discharge from rehabilitation was 94 (range, 56–158) days after aSAH for patients with STX-VCS and 115 (range, 48–327) days for the control group (P=0.16).

Selection of Controls and Matching

Control cases were selected from the period before the introduction of the novel intervention according to the following algorithm:

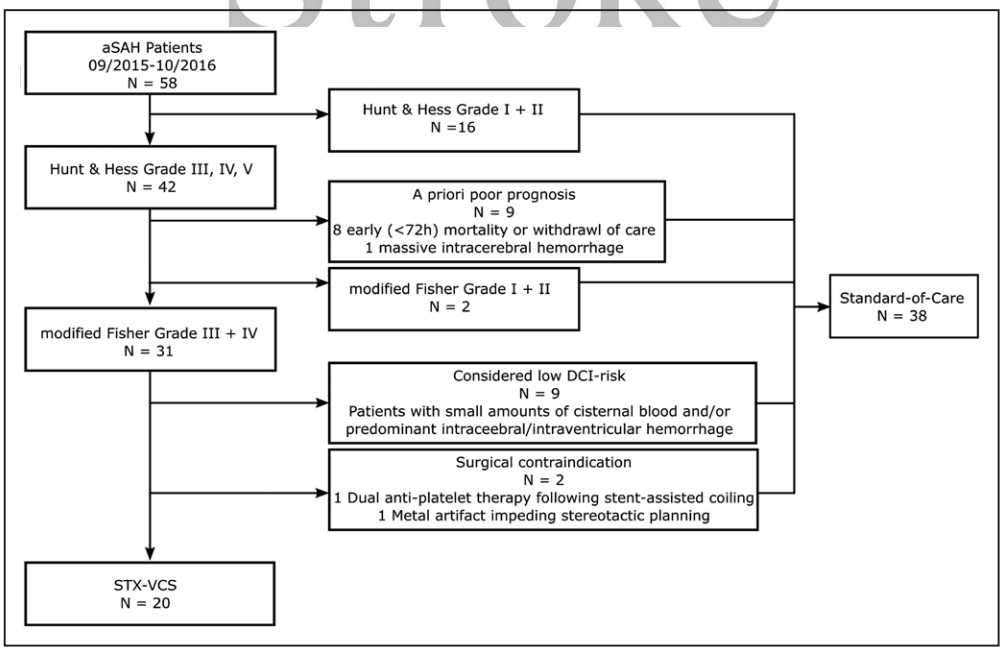

Figure 1. Flowchart: patient selection. The flowchart shows all consecutive aneurysmal subarachnoid hemorrhage (aSAH) patients admitted to our department between September 2015 and October 2016. Selection criteria for stereotactic catheter ventriculocisternostomy (STX-VCS) were admission Hunt and Hess grade ≥3 and modified Fisher grade 3 or 4. Thirty-one patients met these criteria. STX-VCS was not offered in 9 of these patients who were considered at low risk for delayed cerebral infarction (DCI) because of small amounts of cisternal blood. Two patients were ineligible for STX-VCS because of surgical contraindications.

controls were only included if they matched the abovementioned selection criteria for the experimental therapy. Three control patients were assigned to each STX-VCS case (1:3 matching). In backward chronological order (September 2015 toward earlier admission dates), all consecutive aSAH patients admitted to our department were reviewed and included as controls if they met the following matching criteria: age (±12 years), sex, aneurysm treatment method, and admission Hunt and Hess grade. Regarding the Hunt and Hess grade, controls were assigned to STX-VCS cases such that the Hunt and Hess grade of patients with STX-VCS was at least as severe or one point worse as corresponding controls. To render selection of controls more precise, matching patients were only included if their Hijdra sum scores[9] for blood amount and distribution reached a value of ±10 points of corresponding patients with STX-VCS.

To externally validate our results and confirm comparability of cohorts, the likelihood of DCI and expected long term outcomes for both the STX-VCS and the control cohort were estimated by applying current risk assessment scores (de Rooij for DCI[10] and FRESH score [Functional Recovery Expected After Subarachnoid Hemorrhage] for mRS[11]).

Statistical Analyses

DCI was the primary end point of this study. To determine the association between the experimental therapy and DCI, conditional logistic regression was performed using Stata 13 statistical software (Stata statistical software: release 13; StataCorp, College Station, TX). Conditional logistic regression analyses were further performed to assess the association between the experimental therapy and the endpoint mortality and mRS at rehabilitation discharge.

Descriptive statistics of the STX-VCS and standard-of-care cohorts were performed by Fisher exact test, Pearson χ^2 test, or Mann–Whitney *U* test, as appropriate using GraphPad Prism, version 5 (GraphPad Software, San Diego, CA). All reported *P* values are 2 sided, and $P<0.05$ was considered statistically significant.

Results

Between September 2015 and October 2016, 58 consecutive aSAH patients were admitted to our department. Thereof, 20 patients who were—on the basis of case-by-case risk evaluations—considered at high risk for DCI (flowchart, Figure 1) underwent STX-VCS (schematic drawing, Figure 2) followed by fibrinolytic (urokinase) and, in part, spasmolytic (nimodipine) lavage therapy. STX-VCS was performed at a mean 53.8 hours (range, 24–105 hours) after CT diagnosis of aSAH. Precise catheter placement and absence of surgical complications was documented in all patients by postoperative CT. Continuous cisternal lavage >24 hours was feasible in 17 patients (85%). Continuous lavage for >24 hours was not possible because of accidental removal of the external ventricular drain (irrigation outflow tract) in 1 patient. In this patient, the transventricular cisternal catheter was then used as external ventricular drain. In 2 patients, increased intracranial pressure (posterior inferior cerebellar artery infarction secondary to coil occlusion, brain edema because of aSAH) precluded continuous lavage. Start of lavage therapy was associated with 3 episodes of self-limiting asystolia lasting for several seconds in 1 patient. Subsequently, this patient received continuous lavage without adverse effects. Routine CT for blood clearance assessment was not performed. Near-complete subarachnoid blood clearance after initiation of cisternal lavage (Figure 3A and 3C) was observed in 9 patients who presented with an indication for early CT (within 24–48 hours of initiation of cisternal lavage).

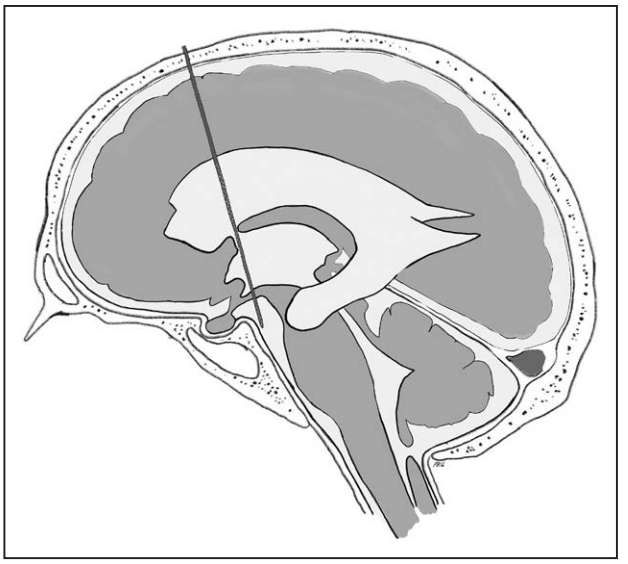

Figure 2. Schematic drawing of stereotactic catheter ventriculocisternostomy. Catheters were implanted via a right or left transfrontal approach passing the lateral ventricle, foramen of Monroi, the third ventricle, perforating its floor. The catheter tip came to lie between the brain stem and dorsum sellae.

Characteristics of patients, aSAH, treatment, and outcome of the 20 patients with STX-VCS and 60 matched controls are summarized in the Table.

The incidence of DCI was significantly higher in the control group (25 of 60 patients, 42%) compared with the patients with STX-VCS (3 of 20 patients, 15%; odds ratio, 0.15; 95% confidence interval, 0.04–0.64). Mortality was 5% in the STX-VCS group versus 33% in the control group (odds ratio, 0.08; 95% confidence interval, 0.01–0.66). Neurological outcome was assessed at discharge from rehabilitation facilities. mRS rating was performed after a median of 94 days for patients with STX-VCS and 115 days for the control cohort ($P=0.16$). Favorable outcome (mRS ≤3) was present in 12 of 20 (60%) STX-VCS and 21 of 60 (35%) controls, respectively. A dichotomized analysis of the mRS (favorable, 0–3 versus unfavorable, 4–6) revealed statistical significance for improved outcome in the STX-VCS group (odds ratio, 0.26; 95% confidence interval, 0.8–0.86).

Transcranial doppler ultrasonography was documented in 55 of 60 (92%) controls and for all patients with STX-VCS for a mean of 12.3±6.0 and 15.4±3.2 days, respectively. Maximum MFV of either proximal intracranial artery >160 cm/s (subcritical vasospasm) were documented in 5 of 20 (25%) patients with STX-VCS and 44 of 55 (80%) controls ($P<0.001$). MFV exceeding 200 cm/s (critical vasospasm) was documented in 4 of 20 (20%) patients with STX-VCS and 30 of 55 (55%) control patients ($P=0.009$). Balloon angioplasty

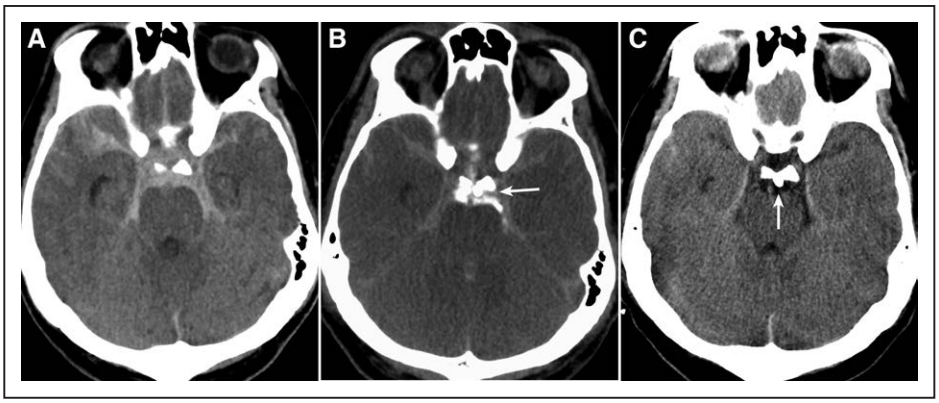

Figure 3. Clearance of subarachnoid blood by cisternal lavage using urokinase. **A**, Computed tomography of a stereotactic catheter ventriculocisternostomy patient before implantation of a cisternal catheter showing classical subarachnoid blood distribution within the basal cisterns and sylvian fissures. **B**, localization of the catheter tip (white arrow, B+C), and distribution of contrast agent (applied via the catheter) within the basal cisterns after catheter placement. **C**, Near-complete resolution of cisternal blood clot after 24 hours of lavage therapy using urokinase.

of both middle cerebral arteries was performed in 1 (5%) STX-VCS patient (on detection of MFV >200 cm/s) in whom increased intracranial pressure precluded spasmolytic lavage therapy. Thirteen (22%) control patients underwent endovascular procedures for the treatment of vasospasm (P=0.17).

According to the treatment concept,[6] all STX-VCS patients with MFV >160 cm/s and eligible for lavage (4 of 5) received cisternal nimodipine (0.005 mg/mL; Bayer Vital GmbH, Leverkusen, Germany) until cessation of vasospasm, at least, however, for 24 hours. MFV was normalized within minutes in 3 patients. Critical MFV unilaterally persisted in 1 patient in whom a peri-insular blood clot persisted, despite fibrinolytic lavage. This patient incurred minor DCI (temporo-parietal cortex, 2.5 cm diameter), despite spasmolytic lavage therapy. Two further patients from the STX-VCS group experienced DCI (1.5 cm, precentral cortex; 5 cm diameter, right temporal cortex) in the absence of sonographic vasospasm. In the control group, DCI affected multiple cerebral perfusion territories in 76% (19/25) and was the cause of mortality in 14 of 18 (78%) patients affected by DCI.

Discussion

DCI is a major source of morbidity and mortality after aSAH. To date, no highly effective treatments are available.[1]

We introduced STX-VCS and lavage therapy as a novel therapeutic option for DCI prevention in patients with aSAH.[6] Otherwise, aSAH patient management was conducted according to international guidelines.[7,12] Importantly, no other changes in aSAH patient management have been adopted in our department in the period concerned. Between September 2015 and October 2016, 20 patients were selected from 58 consecutive aSAH patients (34%) for this treatment on the basis of individual treatment decisions. We selected patients considered at high risk for DCI, that is admission Hunt and Hess grade 3, 4, or 5 and modified Fisher grade 3 or 4 with significant cisternal amounts of blood. We did not approach patients with an a priori dismal prognosis. In this matched cohort study, we compare outcomes with 60 (1:3 matching) historic controls. Controls were taken from the period before the introduction of the experimental procedure to avoid treatment bias. Consecutively (from later to earlier admission dates), all matching controls treated in our department who met the selection criteria for STX-VCS were included to avoid selection bias.

Subarachnoid blood and its break-down products are the putative trigger for DCI. Current treatments for DCI prevention are predominantly symptomatic.[7,12] In contrast, clearance of blood from the subarachnoid space by irrigation and fibrinolytic drugs might represent a causal therapy.[3] The vast majority of cisternal treatment approaches relied on access to the cisterns of the brain during aneurysm surgery.[3] Today, rupturedaneurysms are increasingly secured by endovascular techniques[1,13] (70% in the present series). These patients are not eligible for cisternal treatments because no safe method to access the cisterns of the brain without craniotomy is currently available. To provide access to the cisterns of the brain after aneurysm coiling, catheters have been advanced to the basal cisterns via the lumbar spinal canal.[4,14] A significant reduction in deficits from cerebral vasospasm and improved outcome was reported in a randomized trial by Hamada et al[14] that investigated 2 single urokinase injections (no continuous lavage). However, the use of lumbar catheters resulted in spinal cord injuries in a subsequent study that attempted a continuous lavage therapy via the lumbar spinal canal.[4] The use of lumbar catheters is further limited by the risks of downward herniation in patients with intracranial hypertension, which is frequent after aSAH.

STX-VCS provides a continuous treatment access to the basal cisterns and the ventricles irrespective of the method of aneurysm securing. The stereotactic placement of a transventricular cisternal catheter is challenging but seems to be safe in the setting of severe aSAH. No surgical complications were observed. In our opinion, the high precision of a stereotactic frame-based approach is a key factor for safety of this method. Symptoms of brain stem irritation have to be acknowledged as potential side effects of cisternal lavage using the STX-VCS. Accordingly, initiation of cisternal lavage was associated with short and self-limiting episodes of asystolia in 1 patient. We assume that slow infusion, in particular during initiation of cisternal lavage, may minimize this risk.

Table. Twenty Patients With aSAH Treated by STX-VCS Compared With 60 Matched Controls

	Treatment		Univariate Statistics	Conditional Logistic Regression
	STX-VCS	Standard-of-Care		
Patient characteristics				
No. of patients	20	60		
Year of aSAH, median	2015+2016 (2016)	2008–2015 (2012)		
Sex				
Male	5 (25%)	15 (25%)	P=1.0 (matching variable)	
Female	15 (75%)	45 (75%)		
Age at diagnosis, mean (±SD), y	58.9 (±10.6)	57.8 (±10.6)	P=0.48 (matching variable)	
aSAH characteristics				
Admission Hunt and Hess grade				
III	5 (25%)	14 (23%)	P=0.70; χ^2 (2, N=80) =0.71 (matching variable)	
IV	13 (65%)	43 (72%)		
V	2 (10%)	3 (5%)		
Modified Fisher score				
III	1 (5%)	5 (8%)	P=1.0	
IV	19 (95%)	55 (92%)		
Hijdra-score (mean±SD)	29±7	27±7	P=0.28	
Aneurysm treatment				
Clip	6 (30%)	18 (30%)	P=1.0 (matching variable)	
Coil	14 (70%)	42 (70%)		
Sonographic vasospasm				
Subcritical (MFV >160 cm/s)	5/20 (25%)	44/55 (80%)	P<0.001	
Critical (MFV >200 cm/s)	4/20 (20%)	30/55 (55%)	P=0.009	
Endovascular interventions for vasospasm treatment	1 (5%)	13 (22%)	P=0.17	
Time from aSAH onset to STX-VCS, h, mean±SD	53.8±23.1			
Outcome characteristics				
DCI	3 (15%)	25 (42%)	P=0.03	OR, 0.15; 95% CI, 0.04–0.64
DCI–expected, no. of patients (according to de Rooij score)	11 (54%)	31 (52%)	P=0.44	
Mortality	1 (5%)	20 (33%)	P=0.02	OR, 0.08; 95% CI, 0.01–0.66
Rehabilitation discharge, days after aSAH (median–range)	94 (56–158)	115 (48–327)	P=0.16	
Rehabilitation discharge mRS				
0–3 (independent)	12 (60%)	21 (35%)	P=0.07	OR, 0.26; 95% CI, 0.08–0.86
4–6 (dependent/dead)	8 (40%)	39 (65%)		
mRS expected (according to FRESH score)				
FRESH score (median–range)	4 (3–7)	4 (3–6)	P=0.81	
0–3 (independent)	8 (39%)	24 (40%)	P=1.0	
4–6 (dependent/dead)	12 (61%)	36 (60%)		
Shunt-dependent hydrocephalus in survivors	6/19 (32%)	22/40 (55%)	P=0.11	

aSAH indicates aneurysmal subarachnoid hemorrhage; CI, confidence interval; DCI, delayed cerebral infarction; MFV, mean flow velocity; OR, odds ratio; and STX-VCS, stereotactic catheter ventriculocisternostomy.

Despite methodological limitations,[15] increase of MFV in transcranial doppler is a predictor of DCI after aSAH.[16] We observed significantly less patients affected by MFV increase and shorter duration of pathological MFV in patients who received cisternal lavage. In agreement with previous studies investigating intrathecal treatments after aSAH, vasospasm

and DCI[3] were not entirely eliminated in patients with STX-VCS treatment. This finding corroborates the need for a continuous treatment access, which allows for application of spasmolytic drugs if vasospasm occurs. The use of cisternal nimodipine successfully normalized pathological MFV increase in 3 of 4 patients within minutes.

Because cisternal lavage via STX-VCS is experimental, it was only offered to patients considered at high risk for DCI and mortality. External validity of observed DCI and mortality rates, as well as comparability of both groups analyzed in the present study, was established by current risk scores.[10,11] Accordingly, the expected DCI rate was 54% in the STX-VCS cohort and 52% in the control cohort.[10] The observed DCI incidence in the STX-VCS group was 15%. Further, DCI in the STX-VCS group was minor and unifocal in all 3 cases. Typically, DCI affects multiple vascular territories.[17] In congruence with the literature,[10,17,18] a DCI rate of 42% and lesions in multiple perfusion territories in 76% of patients were observed in the control cohort. Likewise, mortality was in the range of expectations[11] in the control cohort (33%) and significantly lower in patients with STX-VCS (5%).

Favorable long term outcome—as estimated by applying the FRESH score—was expected in 39% of patients with STX-VCS and 40% of the control cohort (P=1.0). Accordingly, favorable neurological outcome at rehabilitation discharge was observed in 35% of the control cohort. By contrast, neurological outcome was improved in STX-VCS patients with 60% showing mRS ≤3 at rehabilitation discharge (odds ratio, 0.26; 95% confidence interval, 0.8–0.86).

Conclusions

The initial results indicate that the incidence of DCI and mortality can be reduced, and outcomes may be improved with this novel intervention. A randomized trial to investigate the efficacy of STX-VCS and lavage therapy is currently being reviewed by regulatory authorities.

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References

1. Macdonald RL, Schweizer TA. Spontaneous subarachnoid haemorrhage. *Lancet*. 2017;389:655–666. doi: 10.1016/S0140-6736(16)30668-7.
2. Macdonald RL. Delayed neurological deterioration after subarachnoid haemorrhage. *Nat Rev Neurol*. 2014;10:44–58. doi: 10.1038/nrneurol.2013.246.
3. Zhang YP, Shields LB, Yao TL, Dashti SR, Shields CB. Intrathecal treatment of cerebral vasospasm. *J Stroke.Cerebrovasc Dis*. 2013;22:1201–1211. doi: 10.1016/j.jstrokecerebrovasdis.2012.04.005.
4. Hänggi D, Eicker S, Beseoglu K, Behr J, Turowski B, Steiger HJ. A multimodal concept in patients after severe aneurysmal subarachnoid hemorrhage: results of a controlled single centre prospective randomized multimodal phase I/II trial on cerebral vasospasm. *Cent Eur Neurosurg*. 2009;70:61–67. doi: 10.1055/s-0028-1087214.
5. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The strengthening the reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. *J Clin Epidemiol*. 2008;61:344–349. doi: 10.1016/j.jclinepi.2007.11.008.
6. Roelz R, Scheiwe C, Urbach H, Coenen VA, Reinacher P. Stereotactic catheter ventriculocisternostomy for clearance of subarachnoid hemorrhage in patients with coiled aneurysms [published online ahead of print June 3, 2017]. *Oper Neurosurg*. https://academic.oup.com/ons/article-abstract/doi/10.1093/ons/opx129/3860939/Stereotactic-Catheter-Ventriculocisternostomy. Accessed June 17, 2017.
7. Steiner T, Juvela S, Unterberg A, Jung C, Forsting M, Rinkel G; European Stroke Organization. European stroke organization guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. *Cerebrovasc Dis*. 2013;35:93–112. doi: 10.1159/000346087.
8. Vergouwen MD, Vermeulen M, van Gijn J, Rinkel GJ, Wijdicks EF, Muizelaar JP, et al. Definition of delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage as an outcome event in clinical trials and observational studies: proposal of a multidisciplinary research group. *Stroke*. 2010;41:2391–2395. doi: 10.1161/STROKEAHA.110.589275.
9. Hijdra A, Brouwers PJ, Vermeulen M, van Gijn J. Grading the amount of blood on computed tomograms after subarachnoid hemorrhage. *Stroke*. 1990;21:1156–1161.
10. de Rooij NK, Greving JP, Rinkel GJ, Frijns CJ. Early prediction of delayed cerebral ischemia after subarachnoid hemorrhage: development and validation of a practical risk chart. *Stroke*. 2013;44:1288–1294. doi: 10.1161/STROKEAHA.113.001125.
11. Witsch J, Frey HP, Patel S, Park S, Lahiri S, Schmidt JM, et al. Prognostication of long-term outcomes after subarachnoid hemorrhage: the FRESH score. *Ann Neurol*. 2016;80:46–58. doi: 10.1002/ana.24675.
12. Connolly ES, Rabinstein AA, Carhuapoma JR, Derdeyn CP, Dion J, Higashida RT, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/american Stroke Association. *Stroke*. 2012;43:1711–1737.
13. Walendy V, Strauss C, Rachinger J, Stang A. Treatment of aneurysmal subarachnoid haemorrhage in Germany: a nationwide analysis of the years 2005-2009. *Neuroepidemiology*. 2014;42:90–97. doi: 10.1159/000355843.
14. Hamada J, Kai Y, Morioka M, Yano S, Mizuno T, Hirano T, et al. Effect on cerebral vasospasm of coil embolization followed by microcatheter intrathecal urokinase infusion into the cisterna magna: a prospective randomized study. *Stroke*. 2003;34:2549–2554. doi: 10.1161/01.STR.0000094731.63690.FF.
15. Washington CW, Zipfel GJ; Participants in the International Multidisciplinary Consensus Conference on the Critical Care Management of Subarachnoid Hemorrhage. Detection and monitoring of vasospasm and delayed cerebral ischemia: a review and assessment of the literature. *Neurocrit. Care.* 2011;15:312–317. doi: 10.1007/s12028-011-9594-8.
16. Kumar G, Shahripour RB, Harrigan MR. Vasospasm on transcranial Doppler is predictive of delayed cerebral ischemia in aneurysmal subarachnoid hemorrhage: a systematic review and meta-analysis. *J Neurosurg*. 2016;124:1257–1264. doi: 10.3171/2015.4.JNS15428.
17. Wong GK, Nung RC, Sitt JC, Mok VC, Wong A, Ho FL, et al. Location, infarct load, and 3-month outcomes of delayed cerebral infarction after aneurysmal subarachnoid hemorrhage. *Stroke*. 2015;46:3099–3104. doi: 10.1161/STROKEAHA.115.010844.
18. Francoeur CL, Mayer SA. Management of delayed cerebral ischemia after subarachnoid hemorrhage. *Crit Care*. 2016;20:277. doi: 10.1186/s13054-016-1447-6.

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