Aneurysmal subarachnoid hemorrhage case series

2023

An observational study included 891 aSAH patients treated at a single center between 2008 and 2018. Data on demography, admission status, radiology, ICP, clinical course, and outcome 1-year post-ictus were collected. Patients treated with thiopental (barbiturate) and decompressive craniectomy were the main target group. Thirty-nine patients (4%) were treated with thiopental alone and 52 (6%) with DC. These patients were younger and had a worse neurological status than those who did not require these treatments. Before thiopental, the median midline shift was 0 mm, whereas basal cisterns were compressed/obliterated in 66%. The median percentage of monitoring time with ICP > 20 mmHg immediately before treatment was 38%, which did not improve after 6 h of infusion. Before DC, the median midline shift was 10 mm, and the median percentage of monitoring time with ICP > 20 mmHg before DC was 56%, which both significantly improved postoperatively. At follow-up, 52% of the patients not given thiopental or operated with DC reached favorable outcomes, whereas this occurred in 10% of the thiopental and DC patients. In summary, 10% of the aSAH cohort required thiopental, DC, or both. Thiopental and decompressive craniectomy are important integrated last-tier treatment options, but careful patient selection is needed due to the risk of saving many patients a state of suffering [1)].

Paavola et al. examined data for 760 consecutive 12-month survivors of aSAH, born in 1950 or after, with a first aSAH from January 1, 1995, to December 31, 2018. Of the 760 patients (median age, 47 years; 53% females; median follow-up, 11 years), 111 (15%) developed epilepsy at a median of 7 months (interquartile range, 2-14 months) after admission for aSAH. Of the 2240 population controls and 4653 first-degree relatives of aSAH patients, 23 (0.9%) and 80 (1.7%) respectively developed epilepsy during the follow-up period. Among the 79 patients with epilepsy in first-degree relatives, 22 (28%) developed epilepsy after aSAH; in contrast, among the 683 patients with no epilepsy in first-degree relatives, 89 (13%) developed epilepsy after aSAH. Having at least one relative with epilepsy was an independent risk factor for epilepsy after aSAH (hazard ratio, 2.44; 95% confidence interval, 1.51-3.95). Cumulative 1-year rates by first-degree relationship were 40% with one or more children with epilepsy, 38% with one or more affected parents, 5% with one or more affected siblings, and 10% with no relatives with epilepsy.

Patients who developed epilepsy after aSAH were significantly more likely to have first-degree relatives with epilepsy than those who did not develop epilepsy after the aSAH [2)].

Multicentre prospective cohort study.

Three central hospitals in Hanoi, Vietnam.

This study included all patients (≥18 years) presenting with aSAH to the three central hospitals within 4 days of ictus, from August 2019 to June 2021, and excluded patients for whom the admission

Glasgow Coma Scale was unable to be scored or patients who became lost at 90 days of follow-up during the study.

The primary outcome was ICH after aneurysm rupture, defined as ICH detected on an admission head CT scan. The secondary outcomes were 90-day poor outcomes and 90-day death.

Of 415 patients, 217 (52.3%) were females, and the median age was 57.0 years (IQR: 48.0-67.0). ICH was present in 20.5% (85/415) of patients with aSAH. There was a significant difference in the 90-day poor outcomes (43.5% (37/85) and 29.1% (96/330); p=0.011) and 90-day mortality (36.5% (31/85) and 20.0% (66/330); p=0.001) between patients who had ICH and patients who did not have ICH. The multivariable regression analysis showed that systolic blood pressure (SBP) ≥140 mm Hg (adjusted odds ratio (AOR): 2.674; 95% CI: 1.372 to 5.214; p=0.004), World Federation of Neurosurgical Societies (WFNS) grades II (AOR: 3.683; 95% CI: 1.250 to 10.858; p=0.018) to V (AOR: 6.912; 95% CI: 2.553 to 18.709; p<0.001) and a ruptured middle cerebral artery (MCA) aneurysm (AOR: 3.717; 95% CI: 1.848 to 7.477; p<0.001) were independently associated with ICH on admission.

Intracerebral hemorrhage was present in a substantial proportion of patients with aneurysmal subarachnoid hemorrhage and contributed significantly to a high rate of poor outcomes and death. Higher systolic blood pressure, worse World Federation of Neurosurgical Societies grading for subarachnoid hemorrhage, and ruptured middle cerebral artery aneurysms were independently associated with ICH on admission [3)]

A retrospective study of patients with aneurysmal subarachnoid hemorrhage who were recruited at two centers, Heidelberg (HD) and Berlin (BE), was performed. Continuous monitoring of mean arterial pressure (MAP) and intracranial pressure (ICP) was recorded. ICP was measured using an intraparenchymal probe in Heidelberg patients and was measure in Berlin patients through external ventricular drainage. Electrocorticographic (ECoG) activity was continuously recorded between 3 and 13 days after hemorrhage. Autoregulation according to Long pressure reactivity index was calculated as a moving linear Pearson's correlation of 20-min averages of MAP and ICP. For every identified Spreading depolarization (SD), 60-min intervals of L-PRx were averaged, plotted, and analyzed depending on SD occurrence. Random L-PRx recording periods without SDs served as the control.

A total of 19 patients (HD n = 14, BE n = 5, mean age 50.4 years, 9 female patients) were monitored for a mean duration of 230.4 h (range 96-360, STD ± 69.6 h), during which ECoG recordings revealed a total number of 277 SDs. Of these, 184 represented a single SD, and 93 SDs presented in clusters. In HD patients, mean L-PRx values were 0.12 (95% confidence interval [CI] 0.11-0.13) during SDs and 0.07 (95% CI 0.06-0.08) during control periods ($p < 0.001$). Similarly, in BE patients, a higher L-PRx value of 0.11 (95% CI 0.11-0.12) was detected during SDs than that during control periods (0.08, 95% CI 0.07-0.09; $p < 0.001$). In a more detailed analysis, CA changes registered through an intraparenchymal probe (HD patients) revealed that clustered SD periods were characterized by signs of more severely impaired CA (L-PRx during SD in clusters: 0.23 [95% CI 0.20-0.25]; single SD: 0.09 [95% CI 0.08-0.10]; control periods: 0.07 [95% CI 0.06-0.08]; $p < 0.001$). This group also showed significant increases in ICP during SDs in clusters compared with single SD and control periods.

Neuromonitoring for simultaneous assessment of cerebrovascular pressure reactivity using 20-min averages of MAP and ICP measured by L-PRx during SD events is feasible. SD occurrence was associated with significant increases in L-PRx values indicative of CA disturbances. An impaired CA was found during SD in clusters when using an intraparenchymal probe. This preliminary study

validates the use of cerebrovascular reactivity indices to evaluate CA disturbances during SDs. Our results warrant further investigation in larger prospective patient cohorts [4].

2022

293 consecutive aneurysmal subarachnoid hemorrhage -patients admitted during a 4-year period was retrospectively analyzed. Cross-sectional muscle measurements were obtained at the level of the third cervical vertebra. Muscle atrophy was defined by a sex-specific cutoff value. Myosteatosis was defined by a BMI-specific cutoff value. Poor neurological outcome was defined as modified Rankin Scale 4-6 at 2 and 6-month follow-up. The patient survival state was checked until January 2021. A generalized estimating equation was performed to assess the effect of muscle atrophy / myosteatosis on poor neurological outcomes after aSAH. Cox regression was performed to analyze the impact of muscle atrophy and myosteatosis on overall survival. The study found that myosteatosis was associated with a poor neurological condition (WFNS 4-5) at admission after adjusting for covariates (odds ratio [OR] 2.01; 95%CI 1.05,3.83; P = .03). It was not associated with overall survival (P = .89) or with poor neurological outcomes (P = .18) when adjusted for other prognostic markers. Muscle atrophy was not associated with overall survival (P = .58) or neurological outcome (P = .32) after aSAH. In conclusion, myosteatosis was found to be associated with the poor physical condition directly after onset of aSAH. Skeletal muscle atrophy and myosteatosis were however irrelevant to outcome in the Western-European aSAH patient. Future studies are needed to validate these finding [5].

2021

A single-center retrospective cohort study was carried out at Trakya University Medical Faculty Training and Practice Hospital. The study includes data on all patients admitted with an aneurysmal subarachnoid hemorrhage between January 1, 2001, and December 31, 2005. Patients were divided into two groups according to their WFNS grade status: Good (I-III) or poor (IV-V) grades. Patients are also classified according to their Glasgow Outcome Scale score: Unfavorable (1-2) or favorable (3-5) outcomes. Data were analyzed statistically, and the effects of the early and ultra-early intervention on the outcome were assessed.

A total of 580 patients were admitted in the study period. Among them, 494 were eligible for the study. The median age (interquartile range) was 55 (18) years. While 244 (49.4%) patients were women, 250 (50.6%) patients were men. Three hundred and fourteen (63.6%) patients were operated on, and 25 patients (5.1%) were undergone endovascular treatment. The ultra-early intervention was achieved in 60 (12.1%) patients and 142 patients (28.7%, including the previous ultra-early intervention group) early intervention was achieved. A meaningful outcome difference was present between the poor-grade ultra-early treatment group and the rest (p=0.007). Analogously, a meaningful outcome difference was present between the poor-grade early treatment group and the rest (p<0.001).

This study supports the growing trend toward early or ultra-early intervention in aneurysmatic subarachnoid hemorrhage. These findings showed that both early and ultra-early interventions have positive effects on the outcome in poor-grade aneurysmatic subarachnoid hemorrhage patients. Future studies with more homogenized and larger samples should be realized to clarify the optimal timing of intervention for aneurysmatic subarachnoid hemorrhage [6].

263 SAH patients were included of which leptin levels were assessed in 24 cases. BMI was recorded along disease severity documented by the Hunt and Hess and modified Fisher scales. The occurrence of clinical or functional DCI (neuromonitoring, CT Perfusion) was assessed. Long-term clinical outcome was documented after 12 months (extended Glasgow outcome scale). A total of 136 (51.7%) patients developed DCI of which 72 (27.4%) developed DCI-related cerebral infarctions. No association between BMI and DCI occurrence (P = .410) or better clinical outcome (P = .643) was identified. Early leptin concentration in serum (P = .258) and CSF (P = .159) showed no predictive value in identifying patients at risk of unfavorable outcomes. However, a significant increase of leptin levels in CSF occurred from 326.0 pg/ml IQR 171.9 prior to DCI development to 579.2 pg/ml IQR 211.9 during ongoing DCI (P = .049). No association between obesity and clinical outcome was detected. After DCI development, leptin levels in CSF increased either by an upsurge of active transport or disruption of the blood-CSF barrier. This trial has been registered at ClinicalTrials.gov (NCT02142166) as part of a larger-scale prospective data collection. BioSAB: https://clinicaltrials.gov/ct2/show/NCT02142166 [7].

2020

A cross-sectional retrospective study was carried out to survey all patients with confirmed aneurysmal subarachnoid hemorrhage operated from March 2011 to September 2016 in an academic vascular center (Rasool Akram Hospital in Tehran, Iran). Of a total of 151 patients, 72 patients were male and 79 were female. The mean age of the participants was 51 years. A transiently CSF diversion (EVD - external ventricular drainage) was performed (the acute hydrocephalus rate) on 21 patients (13.9%). In 36 patients (23.8%), aneurysm occlusion with LTF (lamina terminalis fenestration) and in 115 patients (76.2%) only aneurysm occlusion surgery was performed. In hydrocephalus follow-up after surgery, 13 (12%) patients needed shunt insertion (the rate of shunt-needed hydrocephalus). The statistical analysis demonstrated no significant relation between LTF and shunt-needed hydrocephalus. Confirmation of the hypothesis that LTF may decrease the rate of shunt-needed hydrocephalus can significantly decrease morbidity, mortality, and treatment costs of shunting (that is a simple, but a potentially dangerous procedure). So, it is advised to plan and perform an RCT (randomized controlled trial) that can remove the confounding factors, match the groups, and illustrate the exact effect of LTF on shunt-needed hydrocephalus [8]

Patients were trichotomized into a young cohort (<60 years [n= 268]) and two elderly cohorts (60-65 years [n=60] and ≥65 years [n=77]). The elderly cohorts were analyzed by poor or good scores at presentation (Hunt and Hess [HH] score >3 vs. ≤3, respectively) and poor functional outcome (modified Rankin Scale [mRS] score >2).

Of 137 elderly patients, 121 had a 6-year follow-up. The >65-year-olds (75% [52/69]) were more likely to have poor functional outcomes than the 60-65-year-olds (48% [25/52]) (OR 3.3, 95% CI 1.5-7.1; P=0.002). Among those with an HH score ≤3 at presentation (n=90), the >65-year-old cohort had poorer outcomes than the 60-65-year-old cohort at 6-year follow-up (69% [35/51] vs. 36% [14/39], respectively; OR 3.9, 95% CI 1.6-9.4; P=0.003). Among patients with an HH score >3, no statistically significant differences in functional outcome were observed between the >65-year-old (n=18) and 60-65-year-old (n=13) cohorts.

Aneurysmal subarachnoid hemorrhage outcome in elderly patients are at high risk for poor functional outcomes. However, among those presenting with good HH scores, younger-elderly patients (ages

60-65 years) tend to fare better than older-elderly patients (ages >65 years). Elderly patients presenting with high-grade aSAH fare poorly regardless of age, which can inform clinical decision-making and prognostication [9].

Analysis of 164 aneurysmal subarachnoid hemorrhage survivors (until discharge from intensive care) with the aim to detect factors that influence the length of stay (LOS) in intensive care with multiple linear regression methods. Moreover, binary logistic regression methods were used to examine whether the time in intensive care is a predictor of outcome after 1 year. The clinical 1-year outcome was measured prospectively in a 12-month follow-up by telephone interview and categorized by the modified Rankin Scale (mRS). Patients who died during their stay in intensive care were excluded. Complications like pneumonia (β = 5.11; 95% CI = 1.75-8.46; p = 0.0031), sepsis (β = 9.54; 95% CI = 3.27-15.82; p = 0.0031), hydrocephalus (β = 4.63; 95% CI = 1.82-7.45; p = 0.0014), and delayed cerebral ischemia (DCI) (β = 3.38; 95% CI = 0.19-6.56; p = 0.038) were critical factors depending on the LOS in intensive care as well as decompressive craniectomy (β = 5.02; 95% CI = 1.35-8.70; p = 0.0077). All analyzed comorbidities such as hypertension, diabetes, hypothyroidism, cholesterolemia, and smoking history had no significant impact on the LOS in intensive care. LOS in intensive care (OR = 1.09; 95% CI = 1.03-1.15; p = 0.0023), as well as WFNS grade (OR = 3.72; 95% CI = 2.23-6.21; p < 0.0001) and age (OR = 1.06; 95% CI = 1.02-1.10; p = 0.0061), were significant factors that had an impact on the outcome after 1 year. Complications in intensive care but not comorbidities are associated with higher LOS in intensive care. LOS in intensive care is a modest but significant predictor of outcomes after subarachnoid hemorrhage [10].

2019

Patients with aneurysmal subarachnoid hemorrhage (aSAH) admitted from August 2015 through August 2017 were retrospectively analyzed for EVD placement. Cerebrospinal fluid (CSF) samples were obtained twice weekly for culture and routine studies. Ventriculostomy related infection was defined as the growth of CSF cultures.

During the 2-year study period, 122 patients presented with an aSAH, with 91 (74.6%) having EVD placement. In patients with EVDs, the mean age was 57.9 years (68% female); 88% of aSAHs were Fischer grade III-IV. Mean duration of EVD was 14 days, and 13% of patients required EVD replacement. Endovascular coiling and surgical clipping were performed in 34 (37%) and 53 (58%) patients with EVD, respectively. A total of 347 CSF studies were performed with no EVD-associated infections. There were 3 CSF samples with false-positive Gram stain results but no growth on concurrent or multiple repeat cultures.

Using a standardized protocol for placement and management of EVDs in patients with aSAH is associated with low risk of CSF infection. The study demonstrates that occlusive EVD dressings are not necessary and that routine CSF sampling in patients with EVD may lead to false-positive findings and unnecessary antibiotic administration [11].

Pal'a et al., performed a 2-center, retrospective, clinical database analysis of 732 SAH patients treated between 2008 and 2016. Demographic and clinical data such as age, sex, World Federation of Neurosurgical Societies (WFNS) grade, BMI, Fisher grade, history of arterial hypertension and

smoking, aneurysm location, C-reactive protein (CRP) level, and detailed dosage of vasopressors and nimodipine during the treatment period were evaluated. Clinical outcome was analyzed using the modified Rankin Scale (mRS) 6 months after treatment. Univariate and multivariate regression analyses were performed. Additionally, mean arterial pressure (MAP), age, nimodipine, and vasopressor dose cutoff were evaluated with regard to outcome. The level of significance was set at ≤ 0.05.

Follow-up was assessed for 397 patients, 260 (65.5%) of whom achieved a good outcome (defined as an mRS score of 0-3). Univariate and multivariate analyses confirmed that nimodipine ($p = 0.049$), age ($p = 0.049$), and CRP level ($p = 0.002$) are independent predictors of good outcome. WFNS grade, Fisher score, hypertension, initial hydrocephalus, and total vasopressor dose showed significant influence on outcome in univariate analysis, and patient sex, smoking status, BMI, and MAP showed no significant association with outcome. A subgroup analysis of patients with milder initial SAH (WFNS grades I-III) revealed that initial hydrocephalus ($p = 0.003$) and CRP levels ($p = 0.001$) had significant influence on further outcome. When evaluating only patients with WFNS grade IV or V, age, CRP level ($p = 0.011$), vasopressor dose ($p = 0.030$), and nimodipine dose ($p = 0.049$) were independent predictors of patient outcome. Patients with an MAP < 93 mm Hg, a nimodipine cutoff dose of 241.8 mg, and cutoff total vasopressor dose of 523 mg had better outcomes.

According to the results, higher doses of vasopressors can safely provide a situation in which the maximum dose of nimodipine could be administered. Cutoff values of the total vasopressor dose were more than 3 times higher in patients with severe SAH (WFNS grade IV or V), while the nimodipine cutoff remained similar in patients with mild and severe SAH. Hence, it seems encouraging that a maximum nimodipine dosage can be achieved despite the need for a higher vasopressor dose in patients with SAH [12].

2018

Gas chromatography time-of-flight mass spectrometry was applied to CSF samples collected from 15 consecutive high-grade aSAH patients (modified Fisher grade 3 or 4). Collected CSF samples were analyzed at two time points (admission and the anticipated vasospasm timeframe). Metabolite levels at both time points were compared and correlated with vasospasm status and Glasgow Outcome Scale (GOS) of patients at 1 year post-aSAH. Significance level was defined as $p < 0.05$ with false discovery rate correction for multiple comparisons.

Of 97 metabolites identified, 16 metabolites, primarily free amino acids, significantly changed between the two time points. These changes were magnified in modified Fisher grade 4 compared with grade 3. Six metabolites (2-hydroxyglutarate, tryptophan, glycine, proline, isoleucine, and alanine) correlated with GOS at 1 year post-aSAH independent of vasospasm status. When predicting patients who had low disability (GOS 5 vs. GOS ≤4), 2-hydroxyglutarate had a sensitivity and specificity of 0.89 and 0.83 respectively.

This preliminary study suggests that specific metabolite changes occur in the brain during the course of aSAH and that quantification of specific CSF metabolites may be used to predict long-term outcome in patients with aSAH. This is the first study to implicate 2-hydroxyglutarate, a known marker of tissue hypoxia, in aneurysmal subarachnoid hemorrhage pathogenesis [13].

Thomas et al., from the Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts, hypothesized that the modified Fisher scale is independently associated with cerebrospinal fluid (CSF) macrophage CD163 expression on postictal day 1, and that CSF macrophage CD163 expression is associated with 1-month neurological outcome.

CSF macrophages from 21 SAH and 28 unruptured aneurysm patients (control) were analyzed for CD163 expression using flow cytometry and confocal microscopy on postictal day 1. Significant associations with modified Fisher scale grades or modified Rankin Scale scores were determined using linear regression and a matched case control study.

CSF macrophage CD163 expression was significantly increased in SAH patients compared with controls ($p < 0.001$). The modified Fisher scale (mF) grades ($\beta = 0.407$, $p = 0.005$) and CSF bilirubin concentrations ($\beta = 0.311$, $p = 0.015$) were positively and independently associated with CSF macrophage CD163 expression when the analysis was controlled for age and sex. CSF macrophages from an SAH patient with a high mF grade had increased co-localization of CD163 and glycophorin A (CD235a, an erythrocyte marker) compared with those from an SAH patient with a low mF grade. The controls had no co-localization. CSF macrophage CD163 expression ($p = 0.003$) was inversely associated with 1-month neurological outcome, when SAH patients were matched based on mF grade.

This early study suggests that CSF macrophage CD163 expression, as measured by flow cytometry, may have some neuroprotective function given its inverse association with outcome and provides unique insights into the neuroinflammatory process after SAH [14].

2017

Thirteen patients with SAH with ICH who underwent coil embolization were retrospectively analyzed. Modified Rankin Scale(mRS)scores were compared for postoperative clinical outcomes of different hematoma locations.

All ruptured aneurysms in the present series of patients were treated using endovascular surgery. Six patients underwent additional ventricle drainage. Only one patient underwent craniotomy for evacuation of the hematoma following coil embolization. Despite ten out of thirteen patients(76.9%)having a preoperative SAH clinical grade, as evaluated using the World Federation of Neurosurgical Societies grading system of IV or V, six(46.2%)patients had a favorable outcome(mRS=0-2).

Coil embolization for ruptured aneurysms, especially those located in the frontal lobe, with ICH and without cerebral herniation may be a feasible alternative and less invasive treatment [15].

2016

Mijiti et al, retrospectively reviewed the medical records of 542 consecutive aSAH patients admitted to neurosurgery department of the First Affiliated Hospital of Xinjiang Medical University in Urumqi city of China between January 1, 2011 and December 31, 2015. AV, SV and cerebral infarction were defined based on clinical data and neuroimaging findings. Univariate and multivariate analyses were performed to identify predictors of AV, SV or cerebral infarction.

343 (63.3%) patients fulfilled the inclusion and exclusion criteria. Of them, 182(53.1%) developed AV, 99 (28.9%) developed SV, and 87 (25.4%) developed cerebral infarction. A history of hypertension, poor modified Fisher grade (3-4) and poor Hunt-Hess grade (4-5) on admission were common risk factors for AV, SV and cerebral infarction. Patients from Uyghur ethnic group or other minorities were less likely to develop AV, SV or cerebral infarction, compared to those from Han ethic group after adjustment of other potential confounders. Additionally, age ≥53 years, leukocyte count ≥11× 109/L on admission and being current or former smokers were independent risk factors of cerebral infarction. Leukocyte count ≥11× 109/L on admission and aneurysm size ≥ 10 mm were independent risk factors of SV. Serum glucose level ≥7.0 mmol/L on admission was an independent risk factor of AV.

Risk factors of different definitions of CVS were diverse in Chinese patients with aSAH; however, risk factors of SV and cerebral infarction seem to be similar. We recommend early and aggressive therapy in these patients at-risk of CVS [16].

Sasahara et al., performed plain computed tomography (CT) perfusion (CTP), and CT angiography (CTA) in all patients with aSAH on arrival. Aneurysms were surgically obliterated in patients with stable vital signs and the presence of a brain stem response. They measured the average mean transit time (aMTT) and compared it with the modified Rankin Scale (mRS) score at 1 month. Regions of interest were identified as 24 areas in the bilateral anterior, middle, and posterior cerebral artery territories and 2 areas in the basal ganglia.

A total of 57 patients were treated between 2007 and 2014. None of the 21 patients with aMTT >6.385 seconds achieved a favorable outcome, whereas 8 of the 36 patients with aMTT <6.385 seconds did achieve a favorable outcome (P = 0.015). Furthermore, comparing the number of areas showing a mean transit time (MTT) >7.0 seconds among the aforementioned 8 areas and mRS, favorable outcomes were not seen in 24 patients with more than 2 such areas (P = 0.009).

We cannot expect a favorable outcome for patients with WFNS grade V aSAH with aMTT >6.385 seconds or more than 2 of 8 areas with MTT >7.0 seconds [17].

In a retrospective study, from all 142 adult patients admitted to a surgical intensive care unit (ICU) with SAH between March 2004 and November 2010.

The mean patient age was 54 ± 14 years, 62.7 % were female, and the median Hunt and Hess score was 3. The proportions of patients with poor outcome (Glasgow Outcome Score ≤3) were 58.4, 54.2, and 52.1 % at 3, 6, and 12 months, respectively, after the SAH. The ICU and Hospital mortality rates were both 12.7 %, and the median lengths of stay in the ICU and the hospital were 16 (IQ 7-25) and 26 (IQ 18-34) days, respectively. In multivariable analysis, older age and greater cumulative fluid balance within the first 7 days in the ICU were independently associated with a greater risk of poor outcome.

In this cohort of patients, older age and greater cumulative fluid balance were independently associated with a greater risk of poor outcome up to 1 year after the initial insult. The data suggest that mild hypovolemia may be beneficial in the management of these patients [18].

Seventy-one patients were treated in two periods: 2010-2011 (32 patients; 19 clipped, 6 coiled, 7 untreated), and 2012-2013 (39 patients, 3 clipped, 34 coiled, 2 untreated). No significant differences were found in age, sex, clinical grade at admission, type and location of aneurysm, Fisher score, or in Hospital mortality (28.1% vs 25.6%, P=.35), GOS (except for GOS 5: 43.37% vs 53.8%, P=.045), rate of hydrocephalus and rate of vasospasm. The second cohort obtained better results for aggregated GOS 1+2+3 (36.3% vs 43.75%, P=.034) and for GOS 4+5 (61.5% vs 56.25%, P=.078). The percentage of patients left untreated was significantly lower in the second period (5.1% vs 21.8%, P<.01), as well as the rate of re-bleeding (0% vs 9.4%, P<.01). Patients were treated earlier (2.51 vs 3.95 days), and hospital and total stay were lower (15.2 and 24.6 vs 10.3 and 18 days) in the second period, these differences not reaching statistical significance.

Endovascular therapy allowed treating more patients with aSAH, and with a lower re-bleeding rate. This led to a modest reduction in morbidity and mortality [19].

2015

Five hundred ninety patients (40.4%) reported LOC at onset of SAH. Loss of consciousness was associated with poor clinical grade, more subarachnoid and intraventricular hemorrhage seen on admission computed tomographic scan, and a higher frequency of global cerebral edema (P < .001). Loss of consciousness was also associated with more prehospital tonic-clonic activity (22.7% vs 4.2%; P < .001) and cardiopulmonary arrest (9.7% vs 0.5%, P < .001) vs patients who did not experience LOC. In multivariable analysis, death or severe disability at 12 months was independently associated with LOC after adjusting for established risk factors for poor outcome, including poor admission clinical grade (adjusted odds ratio, 1.94; 95% CI, 1.38-2.72; P < .001). There was no association between LOC at onset and delayed cerebral ischemia or aneurysm rebleeding.

Loss of consciousness at symptom onset is an important manifestation of early brain injury after SAH and a predictor of death or poor functional outcome at 12 months [20].

2006

Between 1999 and 2003, 198 patients with aSAH treated with early aneurysm clipping were analysed. In 1999, a new standardized protocol for intensive care treatment was established in the Department of Neurosurgery, University Hospital Zurich. The results were compared to the earlier time period (1993-1994) immediately after introduction of early aneurysm clipping.

Results: Out of 198 patients with aSAH, 90 patients (45.5%) suffered from mild aSAH World Federation of Neurosurgical Societies (WFNS) grade 1 and 2, 41 (27.3%) from aSAH WFNS grade 3, 36 (18.2%) from grade 4, and 57 (28.8%) from grade 5. From 1999 to 2003, significantly more patients with severe aSAH WFNS grade 4 and 5 underwent (further) treatment (93 out of 198 patients; 47.0%) compared to the former time-period after introduction of early surgery (23 out of 150 patients; 15.3%) (p < 0.0001). In the early series, 10 out of 23 patients (43.5%) with WFNS 4 recovered with good outcome Glasgow Outcome Score 4 and 5, whereas in the later series 23 out of 36 (63.9%) with WFNS grade 4 survived in a good functional state. Before 1999, all patients with WFNS grade 5 died or survived in a vegetative state. From 1999 to 2003, 20 out of 57 patients (35.1%) with aSAH WFNS

grade 5 survived with good outcome.

Conclusions: The availability of extended specialized neurocritical care seems to induce a change within the patient population towards a higher severity grade. Patients with highgrade aSAH might benefit most from highly specialized neurocritical care treatment [21].

1992

The distribution of cisternal blood in relation to the development of acute hydrocephalus was studied in 246 consecutive patients with aneurysmal subarachnoid hemorrhage who were admitted within 72 hours. Patients with evidence on the initial computed tomograph (CT) of subarachnoid hemorrhage caused by other than a ruptured aneurysm and patients with a negative angiography were excluded. Acute hydrocephalus (defined as a bicaudate index, measured on the initial CT or on a repeat CT within 1 week after subarachnoid hemorrhage, exceeding the 95th percentile for age) was found on the initial CT in 50 (20%) of the 246 patients and on a repeat CT in 9 other patients. Ventricular blood was found significantly more often in patients with acute hydrocephalus than in those in whom acute hydrocephalus did not develop (28 of 59 [47%] versus 58 of 187 [31%]; chi 2 = 4.634, p = 0.031). When the analysis was restricted to the 86 patients with ventricular blood, no significant differences were found in the total amount of cisternal blood and in the distribution of cisternal blood between patients with and without hydrocephalus. In contrast, among the 160 patients without ventricular blood, hydrocephalus was associated with a slightly higher total amount of cisternal blood (Wilcoxon's rank sum test, p = 0.023), and significantly more patients with acute hydrocephalus had a higher score in both ambient cisterns than patients without acute hydrocephalus (20 of 31 [65%] versus 41 of 129 [32%]; chi 2 = 10.007, p = 0.002) [22].

1985

Wijdicks et al., studied the sodium balance and changes in plasma volume by an isotope dilution technique in the first week after an aneurysmal subarachnoid hemorrhage in 21 patients. In 11 of the patients, the plasma volume decreased by more than 10%. This was accompanied by a negative sodium balance and hyponatremia in 6 patients, a negative sodium balance without hyponatremia in 4 patients, and a positive sodium balance in 1 patient. Together with a decrease in plasma volume, blood urea nitrogen content increased and body weight decreased. Three patients developed hyponatremia without a decrease in plasma volume. Serum vasopressin was measured in 14 of the 21 patients. The values were elevated on admission and declined in the first week, regardless of the presence of hyponatremia. These findings indicate that natriuresis and hyponatremia in aneurysmal subarachnoid hemorrhage reflect salt wasting and not inappropriate secretion of antidiuretic hormone and that these changes should be corrected by fluid replacement rather than by fluid restriction [23].

1)
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