Aneurysmal subarachnoid hemorrhage case series

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.

Paľa 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.

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 \( \leq 4 \)), 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.

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 \( \geq 53 \) years, leukocyte count \( \geq 11 \times 10^9/L \) on admission and being current or former smokers were independent risk factors of cerebral infarction. Leukocyte count \( \geq 11 \times 10^9/L \) on admission and aneurysm size \( \geq 10 \) mm were independent risk factors of SV. Serum glucose level \( \geq 7.0 \) 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.
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.

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.

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.
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).

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.


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