were queried to identify adult ruptured cAVM patients and subsequently those developing angiographically-confirmed vasospasm. Complex samples multivariable logistic regression and chi-square automatic interaction detection (CHAID) decision tree analyses were performed to identify significant associations between clinical covariates and the development of vasospasm, and a cAVM vasospasm predictive model (cAVM-VPM) was generated based on the effect sizes of these parameters. Results Among 7,215 cAVM patients identified, 935 developed vasospasm, corresponding to an incidence of 13.0%. 110 of these patients (11.8%) subsequently progressed to delayed cerebral ischemia (DCI). Multivariable adjusted modeling identified baseline clinical covariates (decreasing age by decade (aOR 0.87, 95% CI 0.83, 0.92; p < 0.001), female sex (aOR 1.68, 95% CI 1.45, 1.95; p < 0.001), admission Glasgow Coma Scale score < 9 (aOR 1.34, 95% CI 1.01, 1.79; p = 0.045), intraventricular hemorrhage (aOR 1.87, 95% CI 1.17, 2.98; p = 0.009), hypertension (aOR 1.77, 95% CI 1.50, 2.08; p < 0.001), obesity (aOR 0.68, 95% CI 0.55, 0.84; P < 0.001), congestive heart failure (aOR 1.34, 95% CI 1.01, 1.78; p = 0.043), tobacco smoking (aOR 1.48, 95% CI 1.23, 1.78; p < 0.019) and hospitalization events [leukocytosis (aOR 1.64, 95% CI 1.32, 2.04; p < 0.001), hyponatremia (aOR 1.66, 95% CI 1.39, 1.98; p < 0.001), acute hypotension (aOR 1.67, 95% CI 1.31, 2.11; p < 0.001)] independently associated with the development of vasospasm. Intraparenchymal and subarachnoid hemorrhage were not associated with the development of vasospasm following multivariable adjustment. Among significant associations, a CHAID decision tree algorithm identified age 50–59 (parent node), hyponatremia, and leukocytosis as important determinants of vasospasm development. The cAVM-VPM achieved an area under the curve of 0.65 (sensitivity = 0.70, specificity = 0.53). Progression to DCI, but not vasospasm alone, was independently associated with in-hospital mortality (aOR 2.35, 95% CI 1.29, 4.31; p = 0.016) and lower likelihood of routine discharge (to home or to acute rehabilitation) (aOR 0.62, 95% CI 0.41, 0.96; p = 0.031) following adjustment for baseline covariates.

Conclusion This population-based analysis of vasospasm in cAVM identifies common clinical risk factors for its development and establishes progression to DCI as a predictor of poor neurological outcomes.


Q-047 INTERVENTION VERSES OBSERVATION FOR BORDEN TYPE 1 INTRACRANIAL DURAL ARTERIOVENOUS FISTULA: A POOLED ANALYSIS OF 540 PATIENTS

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Background Cerebral cavernous malformations (CCM) are abnormal cystic capillaries with an impaired signaling in endothelial cells originating from venous/capillary bed. They can appear as a sporadic or familial form, caused by somatic and/or inherited loss-of-function mutations in one of risk genes, CCM1–3, or other mutations. They are associated with an elevated risk of focal neurological deficits, seizures and critical hemorrhage following a focal insult.

Aims The aim of this study was to evaluate the effectiveness of endovascular procedures to treat Borden Type 1 intracranial dAVF. This was carried out by conducting a meta-analysis of the published studies of this disease.

Methods A PRISMA guided systematic literature review and meta-analysis was completed to evaluate the outcomes of intervention of any type versus observation alone in the management of low-grade, Borden Type 1 intracranial dAVF. Data from comparative studies were collected from the literature and pooled together with the authors’ own institutional experience. Primary outcome measures included: grade progression, worsening symptoms, death due to dAVF, permanent complications other than death (i.e. from intracerebral hemorrhage or intervention), and rate of death combined with permanent complication. Spontaneous obliteration and procedural complications were also calculated as secondary outcome measures. A fixed effects model was used to calculate pooled odds ratios (OR) for each outcome variable. Rates of non-comparative variables were also calculated.

Results The systematic review yielded 5 comparative studies, that when combined with our experience resulted in a total of 540 patients included in the meta-analysis. The intervention group included 314 patients, while the observation group included 226 patients. There was no significant difference in grade progression (OR: 2.02, 95% CI: 0.51 to 8.03, P = 0.32, I² = 8%), nor in deaths due to dAVF (OR: 3.81, CI: 0.61 to 23.84, P = 0.15, I² = 0%). However, patients within the intervention group had a higher odds of worsening symptoms after intervention (OR: 2.18, CI: 1.08 to 4.43, P = 0.03, I² = 0%). Additionally, intervention was associated with a higher odds of death or permanent complication from either hemorrhage or intervention (OR: 4.35, CI: 1.00 to 19.08, P = 0.05, I² = 0%). Any type of procedural complication occurred in 45/303 (14.9%) patients, with a permanent complication in 9/310 (2.9%) patients. There were no instances (0%) of permanent complications (other than death) due to rupture in either group. Spontaneous obliteration occurred in 24/122 (19.7%) of the observed patients.

Conclusions Pooled analysis of patients with low-grade, Borden Type 1 intracranial dAVF indicates that intervention results in a higher odds of symptom progression, and death or permanent procedural complication. These findings suggest that observation could be favored and patients should be selected for intervention judiciously.


Q-048 PROTEOMICS ANALYSIS ON HUMAN CEREBRAL CAVERNOUS MALFORMATIONS REVEALS NOVEL BIOMARKERS FOR THE DISEASE PATHOLOGY

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Background Cerebral cavernous malformations (CCM) are abnormal cystic capillaries with an impaired signaling in endothelial cells originating from venous/capillary bed. They can appear as a sporadic or familial form, caused by somatic and/or inherited loss-of-function mutations in one of risk genes, CCM1–3, or other mutations. They are associated with an elevated risk of focal neurological deficits, seizures and critical hemorrhage following a focal insult.