SNIS 21st annual meeting oral abstracts

O-001 ELEVATED INTRACRANIAL PCO2 CORRELATES WITH A NEUROPROTECTIVE IMMUNE RESPONSE PROFILE AND IMPROVED NEUROLOGIC OUTCOMES AFTER THROMBECTOMY

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10.1136/jnis-2024-SNIS.1

Introduction Thrombectomy has revolutionized patient outcomes in acute ischemic stroke, but rates of moderate to severe disability remain high. The Perfusion Reconnaissance of the Ischemic Stroke Microenvironment (PRISM) study was designed to analyze perfusion imaging and sampling of both systemic and intracranial blood distal to the thrombus, to better understand dynamic blood gas changes and early proteomic immune and inflammatory response in real time. Among these, carbon dioxide is a known potent vasodilator, potentially improving cerebral blood flow and limiting stroke size and impact.

Methods Patients enrolled in the PRISM study with point-of-care arterial blood gas (ABG) and proteomic analysis (Olink, Boston, MA) were included for analysis. Both ABG and proteomic analysis were assessed intracranially and systemically. Intracranial arterial samples were collected during thrombectomy through the microcatheter prior to stentriever deployment. ABG was analyzed using the iSTAT-1 point-of-care machine (Abbott Laboratories, Chicago, IL) with CG8+ cartridges, measuring pH, pCO_2, pO_2, HCO_3, and base excess. Olink proteomics was used to assess expression of 184 proteins using a proximity extension assay. Outcome measures (NIHSS scores, mRS) were assessed. Statistical analysis included linear regression and Pearson correlation (GraphPad Prism 9.0) with a p value of <0.05.

Results The study involved dual arterial blood samples from 11 patients, with an average age of 64 ± 17.1 years, 9 female participants, average infarct time of 632 ± 323 minutes, NIHSS admit score of 18.1 ± 9.8, and discharge score of 9.7 ± 8.4. Elevated intracranial pCO2 correlated with significantly decreased NIHSS score at discharge ($R^2=0.5546$, $p=0.0055$), which was not observed with systemic pCO2 levels. Further proteomic analysis identified a significant association between elevated intracranial pCO2 levels and increased expression of TNF, TNF-B, TRAIL, TRANCE, IL-12b, and CXCL9 ($p<0.01$), all of which aid in early immune regulation and inflammation response mechanisms. Additionally, elevated intracranial pCO2 was strongly associated with decreased expression of IL-6, LIF, and Caspase-8 ($p<0.01$), key proteins triggering early inflammation and apoptosis.

Conclusion This glimpse into the intracranial intravascular ischemic stroke milieu identifies an early and significant association between elevated intracranial pCO2 and a protective immune response, effectively ameliorating deleterious inflammation and early apoptotic pathways (figure 1 - heatmap demonstrating correlation of intracranial pCO2 and clusters of responsive protein expression). Importantly, similar responses were not observed systemically. This sheds light on the biologic stroke microenvironment, identifying innate pathways leading to better outcomes and potential therapeutic targets to limit secondary injury and enhance stroke recovery.

Disclosures D. Dornbos: 1; C; Siemens Healthineers. 2; C; Imperative Care. **J. Frank:** None. **E. Franz:** None. **N. Millson:** None. **H. Hazelwood:** None. **N. Meredith:** None. **K. Cochran:** None. **M. Cox:** None. **M. Al-Kawaz:** None. **S. Pahwa:** None. **J. Fraser:** None. **K. Pennypacker:** None.

O-002 INFLAMMATORY MARKERS AS PROGNOSTIC PREDICTORS AFTER SUBARACHNOID HEMORRHAGES: A SYSTEMATIC REVIEW AND META-ANALYSIS

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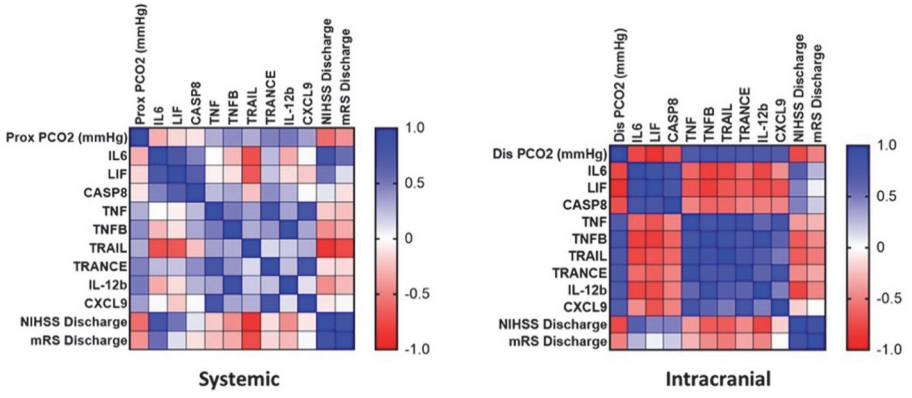
10.1136/jnis-2024-SNIS.2

Subarachnoid hemorrhage (SAH) is an uncommon subtype of stroke that represents a threat to affected patients due to functional subsequent deficits. It is known that multiple inflammatory biomarkers can be altered in this scenario but prognostication of such complications remains unpredictable. We performed a systematic review and meta-analysis to evaluate the prognostic capacity of inflammatory biomarkers in the context SAH. Databases were systematically searched for studies including inflammatory markers in the context of SAH. The main outcomes included delayed cerebral ischemia (DCI) or vasospasm, poor functional outcome, measured by Modified Rankin Scale (mRs) or Glasgow Outcome Scale (GOS), mortality and infection. We included 10532 patients, of whom 2295 were evaluated for c reactive protein (CRP), 2127 were



Abstract O-001 Figure 1

J NeuroIntervent Surg: first published as 10.1136/jnis-2024-SNIS.1 on 21 July 2024. Downloaded from http://jnis.bmj.com/ on September 11, 2024 by guest. Protected by copyright.