

# Global Cerebral Edema in Patients with Subarachnoid Hemorrhage: Correlation with Blood-Brain Barrier Permeability

---

**Abstract No:**

O-30

**Submission Number:**

1069

**Authors:**

J Ivanidze<sup>1</sup>, K Kesavabhotla<sup>1</sup>, D Mir<sup>2</sup>, A Schweitzer<sup>3</sup>, H Baradaran<sup>2</sup>, A Gupta<sup>1</sup>, P Sanelli<sup>4</sup>

**Institutions:**

<sup>1</sup>New York Presbyterian Hospital, Weill Cornell Medical College, New York, NY, <sup>2</sup>Weill Cornell Medical College, New York, NY, <sup>3</sup>New York Presbyterian Hospital, Weill Cornell Medical Center, New York, NY, <sup>4</sup>New York Presbyterian Hospital, Cornell University, New York, NY

**Purpose:**

Global cerebral edema (GCE) in aneurysmal subarachnoid hemorrhage (SAH) is an important predictor of morbidity and mortality. Early detection of GCE remains challenging. Perfusion deficits in SAH patients who develop GCE are thought to be related to impaired autoregulation and blood-brain barrier permeability (BBBP) dysfunction. Computed tomography perfusion (CTP) with full pass technique allows measurement of BBBP in the clinical setting. We assessed whether alterations in BBBP correlate with GCE by studying PS (permeability surface area product), K-trans (flow rate constant from blood plasma to the extravascular extracellular space (EES)), Kep (flow rate constant from EES to blood plasma), and F (blood flow per unit volume of tissue).

**Materials and Methods:**

IRB approval was obtained. Thirty-three SAH patients underwent CTP on admission and at subsequent time points in the early (days 0-3) and delayed phases (days 4-16). Patients were stratified into three outcome groups as GCE, non-GCE and indeterminate based on their admission noncontrast CT using established criteria via blinded consensus reading from two board-certified neuroradiologists. Computed tomography perfusion data were postprocessed into BBBP quantitative maps of PS, K-trans, Kep and Flow using Olea Sphere software (Olea Medical, LaCiotat, France). A total of 2401 ROIs were obtained from the 31 patients included in the analysis. Unpaired t-Tests were performed. In the primary analysis comparing the GCE and non-GCE groups, the indeterminate patients were excluded. A secondary analysis was performed to include indeterminate patients with the GCE group and then the non-GCE group, separately.

**Results:**

A total of 31 patients were included in the statistical analysis; 48.4% (15/31) were classified as GCE, 31.9 % (13/31) as non-GCE and 9.7% (3/31) as indeterminate. The primary analysis revealed that PS, K-trans and F were decreased significantly in GCE compared to non-GCE both in the early and late phases. However, Kep was elevated significantly in GCE compared to non-GCE in both the early and late phases. The secondary analysis demonstrated that the results from the primary analysis are not altered when Indeterminate patients are added to the GCE or non-GCE groups (Figure 1).

**Conclusions:**

Our findings support the hypothesis that altered BBBP function occurs early and remains altered in GCE after SAH. Patients with GCE have significantly decreased PS and Ktrans and significantly elevated KEP, likely due to predominance of flow in the EES to plasma direction given the high volume in the EES. These findings further contribute to our understanding of the pathophysiologic mechanisms underlying GCE.

**Adult Brain:**

New Techniques/Postprocessing

**Adult Brain - Secondary:**

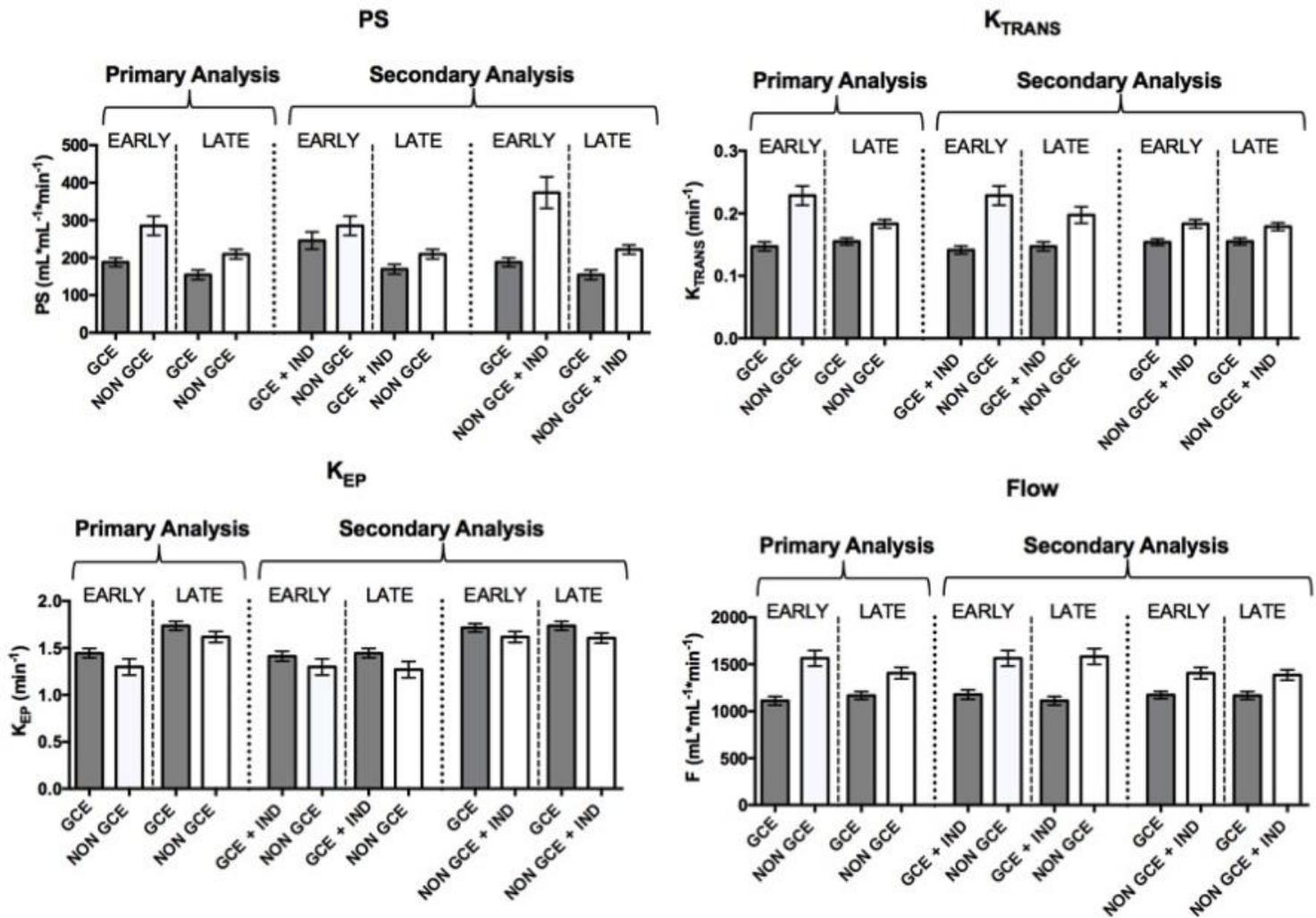
Aneurysms, diagnosis

**Keywords:**

Aneurysmal Subarachnoid Hemorrhage

Cerebral Edema

CT Perfusion



**Reference One:**

Hop JW, Rinkel GJ, Algra A, van Gijn J. Case-fatality rates and functional outcome after subarachnoid hemorrhage: A systematic review. *Stroke; a journal of cerebral circulation.* 1997;28:660-664

**Reference Two:**

Classen J, Carhuapoma JR, Kreiter KT, Du EY, Connolly ES, Mayer SA. Global Cerebral Edema After Subarachnoid Hemorrhage: Frequency, Predictors, and Impact on Outcome . *Stroke .* 2002;33 (5 ):1225–1232.

**Reference Three:**

Mocco J, Prickett CS, Komotar RJ, Connolly ES, Mayer S a. Potential mechanisms and clinical significance of global cerebral edema following aneurysmal subarachnoid hemorrhage. *Neurosurgical focus.* 2007;22(5):E7.

**Reference Four:**

Helbok R, Ko S-B, Schmidt JM, et al. Global cerebral edema and brain metabolism after subarachnoid hemorrhage. *Stroke; a journal of cerebral circulation.* 2011;42(6):1534–9.

**Reference Five:**

Jin X, Liu J, Yang Y, Liu KJ, Yang Y, Liu W. Spatiotemporal evolution of blood brain barrier damage and tissue infarction within the first 3h after ischemia onset. *Neurobiology of disease.* 2012;48:309-316

**Reference Six:**

Kishore S, Ko N, Soares BP, Higashida RT, Tong E, Bhogal S, et al. Perfusion-ct assessment of blood-brain barrier permeability in patients with aneurysmal subarachnoid hemorrhage. *Journal of neuroradiology.* 2012;39:317-325