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RESEARCH-ARTICLE

Blood Degradation Products in the Subarachnoid Space as Predictors of Cerebral Vasospasm and Delayed Cerebral Ischemia Following Aneurysmal Subarachnoid Hemorrhage

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Abstract

Aneurysmal subarachnoid hemorrhage (aSAH) is a critical condition complicated by cerebral vasospasm (CV) and subsequent cerebral infarction (DCIn). One of the key challenges for SAH patient management is predicting and preventing these secondary complications, because many SAH patients survive the initial hemorrhage and possibly could be treated if these secondary complications were identified on time. While current clinical methods can assess SAH severity and patient outcomes, their use in predicting CV and DCI is limited. Emerging evidence suggests that blood degradation products (BDPs) resulting from the breakdown of leaked blood in the subarachnoid space (SAS) may play a significant role in triggering CV and DCI. The results of this study support the hypothesis that simulation of blood degradation products transport can be used to predict CVs and subsequent DCIn for aSAH patients.

CCS Concepts

• **Applied computing**; • **Life and medical sciences**; • **Health informatics**;

Keywords

Subarachnoid hemorrhage (SAH), cerebral vasospasm (CV), delayed cerebral ischemia (DCI), blood degradation products (BDPs), computational fluid dynamics (CFD)

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1 Introduction

Aneurysmal subarachnoid hemorrhage (aSAH) is a life-threatening condition associated with high mortality (32-67%) and significant long-term morbidity [1]. Although most patients survive the hyperacute phase, they remain at risk for complications such as large-vessel vasospasm (LVV), small vessel vasospasm (SVV) or in general cerebral vasospasm (CV), which contributes to delayed cerebral ischemia (DCI) and permanent infarction (DCIn).

Cerebral vasospasm is a common and potentially treatable phenomenon following SAH, characterized by the narrowing of large and medium-sized intracranial arteries. Another potentially treatable phenomenon following SAH is delayed cerebral ischemia (DCI), which is a clinical syndrome of focal neurological and/or cognitive deficits.

While strong statistical and territorial correlations exist between LVV and DCIn, clinical interventions targeting vasospasm have not consistently improved outcomes [2]—possibly due to suboptimal intervention timing.

To test this, we developed a numerical model that incorporates patient-specific data from routine clinical tests and provides blood degradation products (BDPs) distribution in the subarachnoid space (SAS).

2 Methods

This study was approved by the Vilnius Regional Biomedical Research Ethics Committees (Protocol No. 2021/9-1370-847) and conducted in accordance with the Declaration of Helsinki. Informed

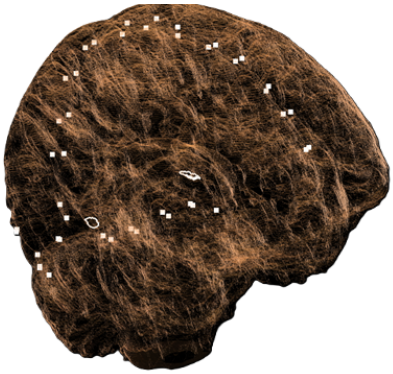

Figure 1: "Model Brain" where the CSF flow and BDP transport is simulated. White points depict the outflow locations and white contours depict the inflow locations.

consent was obtained from all participants regarding the participation and publication of any images, clinical data, or any other data included in the manuscript. The study adheres to all regulations and guidelines involving human research.

A total of 15 aSAH patients (mean age 59±13 years) underwent standard imaging (NCCT and CTA) at Vilnius University Hospital Santaros Clinics (Sept 2021–Sept 2023). A "Model Brain" for simulations was created from high-quality MRI T2-weighted scan of a healthy 42-year-old male. The Slicer3D was used for the cranial cerebrospinal fluid space (cCSFS) volume segmentation, which incorporated cerebral ventricles, SAS, and periarterial spaces around arteries segmented from MRI-angiography scan. Patient-specific hemorrhages were mapped onto the "Model Brain", while BDPs transport via CSF was simulated using computational fluid dynamics with COMSOL Multiphysics. The Model Brain with depicted boundary conditions is shown in Figure 1.

3 Results

The numerical simulations allowed obtaining BDP concentrations inside the "Model Brain" for each patient. For locality assessment purposes, the "Model Brain" was segmented into six basins based on the arterial blood supply, including the left (sin) and right (dex) sides of the anterior cerebral artery (AcA) basin, middle cerebral artery (AcM) basin, and posterior cerebral artery (AcP) basin.

A total of 90 events were analyzed (15 patients ∗ 6 territories), of which 27 (30%) exhibited LVV and 17 (18.9%) developed DCIn. Notably, every infarcted territory was associated with an LVV event (Table 1), and it had ~63% chance of DCIn event, and ~37% chance of no DCIn, when LVV was present, and 0% chance of DCIn when no LVV event was present. Fisher's exact test confirmed a statistically

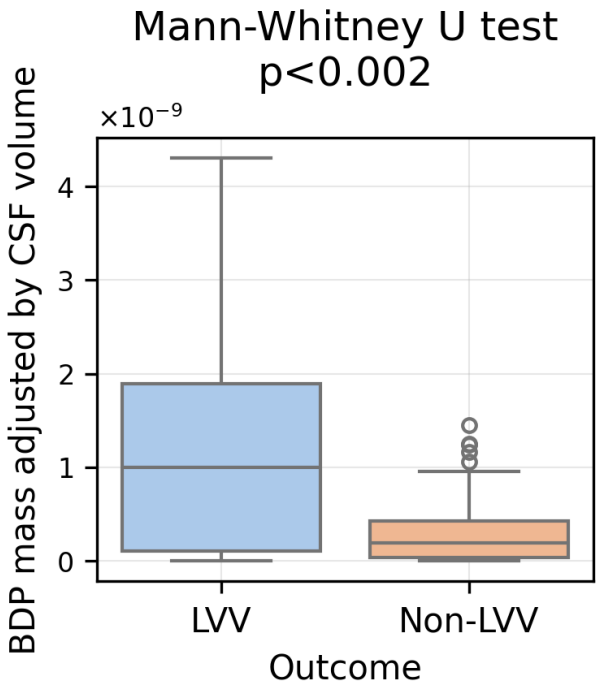

Figure 2: The Mann-Whitney U test between patient's CSF volume adjusted BDP mass in each major arterial territory and patient outcome in terms of LVV.

significant association between LVV and DCIn ($p < 0.001$), which is consistent with [3].

The Mann-Whitney U test confirmed a statistically significant difference in BDP mass between LVV and non-LVV groups ($p < 0.002$) shown in Figure 2. These findings suggest that higher BDP concentrations in the subarachnoid space, adjusted by CSF volume, are associated with an increased risk of LVV and subsequently increased risk of DCIn.

4 Limitations

The number of patients included in the analysis (15) is not sufficient to draw general conclusions. To validate these findings a larger patient cohort would be necessary.

The segmentation of Model Brain into six basins for the locality analysis could still be too coarse to capture very localized events, as cerebrovascular anatomy is much more complex. Analysis on the spatial distribution of BDPs at all Model Brain points could provide more detailed insights into localized risk for LVV, and DCIn. The

Table 1: LVV and DCIn events in major cerebral arterial territories

Events	DCIn	Non-DCIn
LVV	17	10
Non-LVV	0	63

peak BDP concentrations in subarachnoid space may have a strong local effect for LVV, and DCIn development.

5 Conclusions

Computational fluid dynamics simulation allows obtaining BDP concentration inside the detailed SAS, which demonstrates its potential to be the predictive marker for CV and DCI events following SAH. A statistically significant difference in BDP mass between LVV and non-LVV groups ($p < 0.002$) was obtained, which supports the hypothesis that simulation of BDPs transport can be used to predict LVVs for aSAH patients. Early prediction of LVVs could enable timely, targeted interventions and improve aSAH patient outcomes.

Acknowledgments

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