Aneurysmal subarachnoid hemorrhage biomarkers

Subarachnoid hemorrhage (SAH), particularly aneurysmal subarachnoid hemorrhage (aSAH), is a serious cerebrovascular condition that often requires rapid diagnosis and treatment. Biomarkers can help in the early diagnosis, prognosis, and management of SAH by reflecting brain injury, inflammation, or vascular pathology. Here are some key biomarkers for SAH:

1. **Inflammatory Cytokines**:

1. **Interleukin-1β (IL-1β)**: Elevated levels of IL-1β are associated with increased inflammation and can worsen brain injury following SAH.
2. **Interleukin-6 (IL-6)**: High IL-6 levels in cerebrospinal fluid (CSF) and blood are linked with worse outcomes in SAH patients and can predict cerebral vasospasm.
3. **Tumor Necrosis Factor-alpha (TNF-α)**: TNF-α is involved in neuroinflammatory responses post-SAH and may be correlated with the severity of brain injury.

2. **Oxidative Stress Markers**:

1. **Malondialdehyde (MDA)**: MDA is a marker of lipid peroxidation and oxidative stress, which is increased in SAH due to secondary injury mechanisms.
2. **8-Isoprostane**: This is an indicator of lipid peroxidation and oxidative damage, particularly in the early stages of SAH. It has been associated with increased risk of vasospasm and delayed cerebral ischemia.

3. **Neurofilament Light Chain (NFL)**:

1. NFL is a marker of neuronal injury, detectable in blood and CSF, and is associated with the severity of brain injury in SAH. Elevated levels may indicate axonal damage and correlate with poor neurological outcomes.

4. **Matrix Metalloproteinases (MMPs)**:

1. **MMP-9 and MMP-2**: These enzymes degrade extracellular matrix components, which can lead to blood-brain barrier breakdown and inflammation. High MMP levels in the CSF post-SAH may indicate increased risk of cerebral edema and vasospasm.

5. **Endothelin-1 (ET-1)**:

1. ET-1 is a potent vasoconstrictor linked with cerebral vasospasm, a common complication of SAH. High ET-1 levels are associated with increased risk of vasospasm and may help in risk stratification for delayed cerebral ischemia.

6. **S100B**:

1. S100B is a calcium-binding protein produced by astrocytes and reflects blood-brain barrier disruption and astrocyte activation. Elevated S100B levels in blood and CSF have been associated with SAH severity, cerebral edema, and poor prognosis.

7. **Neuron-Specific Enolase (NSE)**:

1. NSE is released by neurons during brain injury, and increased levels in blood or CSF may indicate neuronal damage. High NSE levels are generally associated with poor neurological outcomes in SAH patients.

8. **Glial Fibrillary Acidic Protein (GFAP)**:

1. GFAP is released from astrocytes during brain injury and blood-brain barrier disruption. Elevated GFAP levels in blood or CSF after SAH are correlated with brain injury severity and have been proposed as a biomarker for cerebral edema and poor outcomes.

9. **Hemoglobin Degradation Products**:

1. **Bilirubin Oxidation Products**: Elevated levels can indicate subarachnoid blood breakdown and correlate with the severity of hemorrhage and vasospasm risk.
2. **Haptoglobin and Ferritin**: These proteins are involved in hemoglobin scavenging and are elevated following SAH, often associated with oxidative stress and inflammation.

10. **Endothelial and Platelet Markers**:

1. **Von Willebrand Factor (vWF)**: A marker of endothelial damage, vWF is often elevated in SAH and can correlate with blood-brain barrier dysfunction and risk of vasospasm.
2. **Soluble P-Selectin and E-Selectin**: These are markers of platelet activation and endothelial cell damage, which can indicate inflammation and microvascular thrombosis post-SAH.

Conclusion

The identification of biomarkers for SAH is evolving, with each providing different insights into the pathophysiology, prognosis, and treatment response. Some, like IL-6 and ET-1, are closely linked with complications such as vasospasm, while others, like S100B and GFAP, are indicative of the degree of brain injury and prognosis. Using these biomarkers in combination may improve diagnostic accuracy, guide treatment strategies, and help monitor progression and outcomes in SAH patients.

IL-1β on the Development of Aneurysmal Subarachnoid Hemorrhage

A study aimed to investigate the effect and clinical implications of IL-1β on the development of aneurysmal subarachnoid hemorrhage.

This retrospective study included a total of 80 participants, and these participants were divided into the following two groups: control group (healthy participants) and experimental group (aneurysmal subarachnoid hemorrhage patients). Then, all of the participants received digital subtraction angiography or computed tomography angiography. Participants' general data were collected and analyzed. IL-1β expression in blood samples were determined by ELISA, and then IL-1β protein were determined by western blotting.

A total of 80 participants was included in this study, and the participants` general data, including gender, age, and previous medical history, showed no significant differences between the experimental group and control group. The IL-1β value in the experimental group was significantly increased, and the difference was statistically significant ($p < 0.05$).

Upregulated IL-1β can promote the development of aneurysmal subarachnoid hemorrhage, indicating that IL-1β is a key factor in evaluating the prognosis of patients with aneurysmal subarachnoid hemorrhage [1].

1)

Li F, Zhang W, Wang M, Jia P. The Effect and Clinical Implications of IL-1β on the Development of Aneurysmal Subarachnoid Hemorrhage. Clin Lab. 2024 Nov 1;70(11). doi: 10.7754/Clin.Lab.2024.240608. PMID: 39506594.

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