Intracerebral hemorrhage complications

- Growth Factors and the Choroid Plexus: Their Role in Posthemorrhagic Hydrocephalus
- Perimesencephalic Subarachnoid Hemorrhage Bleeding Patterns Are Not Always Benign: Prognostic Impact of an Aneurysmal Pathology
- Japanese Nationwide Questionnaire Survey on the Treatment and Management of Subarachnoid Hemorrhage Due to Ruptured Cerebral Aneurysm
- Regional Differences in Presentation, Cause, and Outcome of Reversible Cerebral Vasoconstriction Syndrome
- Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage: a narrative review
- Embolic Shower Pattern on Magnetic Resonance Imaging: An Atypical Presentation of Chikungunya Encephalitis
- Anesthetic and perioperative management of pregnant patients undergoing neurosurgery: a case series from a single center in Morocco (2017-2024)
- Sympathetic nerve block as an add-on therapy for intervention and prevention of cerebral vasospasm after subarachnoid hemorrhage

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Intracerebral hemorrhage complications can be severe and may have long-term consequences. Some of the potential complications include:

Intracranial hypertension (ICP): The accumulation of blood within the brain can lead to an increase in intracranial pressure. Elevated ICP can compress brain structures, impair blood flow, and potentially cause brain herniation, a life-threatening condition.

Brain edema, or swelling of brain tissue, often accompanies ICH. Edema can worsen the initial neurological symptoms and increase intracranial pressure, making it crucial to monitor and manage.

Mass Effect: As the hematoma (pool of blood) enlarges, it can compress nearby brain tissue, leading to neurological deficits, such as weakness, numbness, or loss of consciousness.

Brain herniation syndromes: Severe ICH can lead to brain herniation, where brain tissue is pushed or squeezed into areas of the brain where it doesn't belong. Herniation can be life-threatening and result in loss of vital functions.

Vasospasm: After ICH, blood vessels in the surrounding areas may go into spasm, narrowing the vessels and reducing blood flow. This can lead to further brain injury or ischemia.

Infections: There is a risk of infection when the protective barrier around the brain is compromised due to ICH. Meningitis or brain abscesses can develop and require prompt treatment with antibiotics or surgical intervention.

see Brain abscess after intracerebral hemorrhage.

Sepsis [1)]

Cerebral contusion (CC) results in a release of catecholamines, autonomic dysfunction, and neural stimulation that can lead to a number of cardiac adverse events, so it is critical to determine these. ECG changes on admission showing a prolonged corrected QT interval have prognostic significance in CC. This simple and easily applicable information should be taken into consideration at the time of clinical decision making which may prevent an adverse events survivor [2)]

Cognitive and Functional Impairments: Depending on the location and extent of the hemorrhage, ICH can lead to cognitive deficits, speech problems, difficulty with motor skills, and other functional impairments. Rehabilitation is often needed to help patients regain lost functions.

Emotional and Psychological Effects: The emotional trauma associated with ICH can lead to depression, anxiety, or post-traumatic stress disorder (PTSD). Psychological support and therapy may be necessary.

Intracerebral hemorrhage recurrence: In some cases, individuals who have experienced an ICH may be at increased risk of a recurrent hemorrhage, particularly if underlying conditions like hypertension are not adequately managed.

The prognosis and risk of complications in ICH depend on several factors, including the location and size of the hemorrhage, the patient's overall health, and the promptness and effectiveness of medical treatment. s.

Prior reports have identified that radiographic factors such as the presence of a subdural hematoma (SDH) subarachnoid hemorrhage (SAH), skull fracture, and larger contusion volume are associated with higher risk for HPC, as well as clinical factors such as older age, hyperglycemia, antiplatelet medication use, low platelet count, and timing of the initial CT scan [3)].

Inflammatory response mediates secondary brain injury during intracerebral hemorrhage (ICH).

Porencephaly, a cystic lesion lined with connective tissue or glial tissue that may communicate with the ventricular system, often caused by vascular infarcts or following intracerebral hemorrhage or penetrating trauma (including repeated ventricular punctures).

Stress ulcer.

Hematoma expansion

see Hematoma expansion.

Hematoma expansion is an important determinant of outcome in spontaneous intracerebral hemorrhage (ICH) due to small vessel disease (SVD).

Approximately 15% of patients experience seizures after spontaneous intracerebral hemorrhage (ICH). The pathogenesis of seizures post-ICH is not well-known; however, iron deposition-related neuronal injury following hemoglobin breakdown may contribute. Profiling known MicroRNAs to identify biomarkers for post-ICH late seizures, they found 64 differentially expressed MicroRNA: 32 upregulated and 32 downregulated in seizure vs. non-seizure. Functional classification of upregulated MicroRNA for KEGG pathways and biological processes identified enrichment for cell cycle, protein modifications, and FoxO neurotrophin signaling pathways. No significant enrichment was found for downregulated MicroRNA. Molecular functions Gene Ontology (GO) terms enriched for upregulated MicroRNA are numerous, while downregulated MicroRNAs were associated with ion channel activity. RT-PCR confirmed two MicroRNAs, 4317 and 4325, were differentially expressed in patients who developed seizures at 1 year. MiR-4317 regulates SLC38A1, a glutamine-glutamate transporter. Integrated MicroRNA-mRNA network analysis identified COMMD6, APOBEC2, and RASSF6-involved in NF-kB regulation. Two MicroRNAs (miR-4317 and 4325) differentiated post-ICH late seizures vs. non-seizures at 1 year. The results suggest functional and MicroRNA-mRNA networks as potential biomarkers for post-ICH late seizures [4].

Hydrocephalus

In some cases, ICH can block the flow of cerebrospinal fluid (CSF), leading to hydrocephalus. This can cause an accumulation of CSF and increased intracranial pressure, requiring treatment.

Ischemia

Ischemia on diffusion-weighted imaging (DWI) are common after acute spontaneous intracerebral hemorrhage (ICH) but are poorly understood for large ICH volumes (> 30 mL). Rivera-Lara et al. hypothesized that large blood pressure drops and effect modification by cerebral small vessel disease markers on magnetic resonance imaging (MRI) are associated with DWI lesions.

This was an exploratory analysis of participants in the Minimally Invasive Surgery Plus Alteplase for Intracerebral Hemorrhage Evacuation phase 3 trial with protocolized brain MRI scans within 7 days from ICH. Multivariable logistic regression analysis was performed to assess biologically relevant factors associated with DWI lesions, and relationships between DWI lesions and favorable ICH outcomes (modified Rankin Scale 0-3).

Of 499 enrolled patients, 300 had MRI at a median of 7.5 days (interquartile range 7-8), and 178 (59%) had DWI lesions. The incidence of DWI lesions was higher in patients with systolic blood pressure (SBP) reduction ≥ 80 mm Hg in the first 24 h (76%). In adjusted models, factors associated with DWI lesions were as follows: admission intraventricular hematoma volume ($p = 0.03$), decrease in SBP ≥ 80 mm Hg from admission to day 1 ($p = 0.03$), and moderate-to-severe white matter disease ($p = 0.01$). Patients with DWI lesions had higher odds of severe disability at 1 month ($p = 0.04$), 6

months (p = 0.036), and 12 months (p < 0.01). No evidence of effect modification by cerebral small vessel disease on blood pressure was found.

In patients with large hypertensive intracerebral Hemorrhage, white matter disease, intraventricular hemorrhage volume and large reductions in SBP over the first 24 h were independently associated with DWI lesions. Further investigation of potential hemodynamic mechanisms of ischemic injury after large intracerebral Hemorrhage is warranted [5)]

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Lin J, He M, Tan B, Yang X, Li Y, Xian J, Feng H, Chen Y. Impact and risk factors of sepsis on long-term outcomes after spontaneous intracerebral hemorrhage. Chin Med J (Engl). 2022 Feb 16. doi: 10.1097/CM9.0000000000001954. Epub ahead of print. PMID: 35170514.
2)
Yavuz AY, Baskurt O, Kurtulus Y, Avci I. Prognostic significance of prolonged corrected QT interval in cerebral contusion. Indian J Med Res. 2023 Sep 14. doi: 10.4103/ijmr.ijmr_3629_21. Epub ahead of print. PMID: 37706372.
3)
Allison RZ, Nakagawa K, Hayashi M, Donovan DJ, Koenig MA. Derivation of a Predictive Score for Hemorrhagic Progression of Cerebral Contusions in Moderate and Severe Traumatic Brain Injury. Neurocrit Care. 2017 Feb;26(1):80-86. doi: 10.1007/s12028-016-0303-5. PubMed PMID: 27473209; PubMed Central PMCID: PMC5233574.
4)
Iwuchukwu I, Nguyen D, Beavers M, Tran V, Sulaiman W, Fannin E, Lasseigne L, Ramsay E, Wilson J, Bazan NG. MicroRNA Regulatory Network as Biomarkers of Late Seizure in Patients with Spontaneous Intracerebral Hemorrhage. Mol Neurobiol. 2020 Feb 10. doi: 10.1007/s12035-020-01872-y. [Epub ahead of print] PubMed PMID: 32040835.
5)
Rivera-Lara L, Cho SM, Li Y, Ali H, McBee N, Awad IA, Avadhani R, Hanley DF, Gandhi D, Walborn N, Murthy SB, Ziai WC. Mechanistic Evaluation of Diffusion Weighted Hyperintense Lesions After Large Spontaneous Intracerebral Hemorrhage: A Subgroup Analysis of MISTIE III. Neurocrit Care. 2023 Dec 1. doi: 10.1007/s12028-023-01890-3. Epub ahead of print. PMID: 38040993.