Aneurysmal subarachnoid hemorrhage prognosis

J.Sales-Llopis

Neurosurgery Department, Alicante University General Hospital, Alicante, Spain.

- Does duration of nimodipine therapy impact outcome in aneurysmal subarachnoid hemorrhage: systematic review and meta-analysis
- Perimesencephalic Subarachnoid Hemorrhage Bleeding Patterns Are Not Always Benign: Prognostic Impact of an Aneurysmal Pathology
- Role of Magnetic Resonance Venography in the Evaluation of Cerebral Veins and Sinuses Occlusion
- Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage: a narrative review
- Comprehensive predictive modeling in subarachnoid hemorrhage: integrating radiomics and clinical variables
- Post-traumatic hydrocephalus after decompressive craniectomy: a multidimensional analysis of clinical, radiological, and surgical risk factors
- Risk factors for the development of hydrocephalus in traumatic brain injury: a systematic review and meta-analysis
- Analysis of short-term efficacy and rebleeding risk in aneurysmal subarachnoid hemorrhage patients undergoing vascular intervention

---

A study concludes that elevated Stress Hyperglycemia Ratio (SHR) is significantly associated with poor functional prognosis at one year in patients with aSAH, independent of pre-existing diabetes. This highlights the importance of managing acute hyperglycemia in the setting of neurological injury to potentially improve outcomes. The results suggest that SHR could serve as a valuable prognostic marker in patients with aSAH, helping to identify those at higher risk of poor outcomes and potentially guiding therapeutic strategies to mitigate these risks [1)]

Test First

Introduction: 1. Aneurysmal subarachnoid hemorrhage (aSAH) is a devastating form of stroke affecting which population?

1. [ ] Elderly individuals
2. [ ] Pediatric patients
3. [X] Working-age population
4. [ ] Geriatric population

2. What percentage of all strokes does aSAH account for?

1. [ ] 1%
2. [X] 5%
3. [ ] 10%
4. [ ] 20%

3. Which of the following is a common complication and major prognostic factor for increased morbidity in aSAH survivors?

1. [X] Epilepsy
2. [ ] Aneurysm rebleeding
3. [ ] Cerebral vasospasm
4. [ ] Intracerebral hematoma

4. Cerebral vasospasm and delayed cerebral ischemia occur in what percentage of aSAH patients?

1. [ ] 10-20%
2. [ ] 30-40%
3. [X] 50-70%
4. [ ] 80-90%

5. What is the gold standard imaging technique for detecting vasospasms of the vessels of the circle of Willis?

1. [ ] Transcranial Doppler ultrasonography (TCD)
2. [ ] Computed tomography (CT)
3. [ ] Magnetic resonance imaging (MRI)
4. [X] Digital subtraction angiography (DSA)

6. Which factor is strongly associated with delayed cerebral ischemia (DCI) and clinical outcome in aSAH patients?

1. [ ] Aneurysm rebleeding
2. [ ] Hypertension
3. [X] Angiographic vasospasm
4. [ ] Microcirculation disturbances

7. What is the case fatality rate of aneurysmal subarachnoid hemorrhage (aSAH) due to the initial hemorrhage or subsequent complications?

1. [ ] 25%
2. [X] 50%
3. [ ] 75%
4. [ ] 100%

8. What is the second leading cause of disability after the initial hemorrhage in aSAH patients?

1. [X] Cerebral vasospasm
2. [ ] Intracerebral hematoma
3. [ ] Epilepsy
4. [ ] Infection

9. What is the primary aim when managing aSAH patients with aneurysms?

1. [ ] Reducing inflammation
2. [ ] Preventing microcirculation disturbances
3. [X] Occlusion of the aneurysm
4. [ ] Minimizing intracerebral hematoma

10. Which patient group is at a higher risk of experiencing complications following aSAH?

1. [ ] Pediatric patients
2. [X] Elderly patients
3. [ ] Middle-aged adults
4. [ ] Adolescents

Abstract

Aneurysmal subarachnoid hemorrhage (aSAH) is a devastating form of stroke that disproportionately affects the working-age population. This condition is marked by the rupture of an intracranial aneurysm, leading to the release of blood into the subarachnoid space, which can have severe consequences. Among the many complications that can arise from aSAH, epilepsy stands out as a common and significant factor influencing patient prognosis.

This article provides an overview of the multifaceted challenges associated with aSAH, including its impact on the working-age population and the role of epilepsy as a major prognostic factor for increased morbidity in survivors. Additionally, the article highlights the importance of understanding the complications and outcomes related to aSAH, which may provide insights into the management and treatment of this critical medical condition.

The article also touches on the potential influence of factors such as neutrophil-to-albumin ratio, genetic polymorphisms, and inflammatory markers, which can affect the clinical course and outcomes in aSAH. By addressing these various components and their interplay, healthcare professionals and researchers aim to improve the prediction, prevention, and management of aSAH, ultimately leading to better outcomes and quality of life for affected individuals.

Introduction

Aneurysmal subarachnoid hemorrhage (aSAH) is a devastating form of stroke affecting the working-age population, where epilepsy is a common complication and major prognostic factor for increased morbidity in aSAH survivors.

Aneurysmal subarachnoid hemorrhage (aSAH) occurs in about 5% of all strokes and has still a mortality of 50% and a significant morbidity in survivors [2].

The second cause of disability after the initial hemorrhage is cerebral vasospasm and the delayed cerebral ischemia which occurs in 50–70% of patients [3].

These two pathological entities seem to have different pathophysiological etiologies and cannot be detected by the same techniques. Vasospasms of the vessels of the circle of Willis can be detected by transcranial Doppler ultrasonography (TCD), whereas microcirculation disturbances can be detected by perfusion imaging techniques. Digital subtraction angiography (DSA) remains until now the gold

standard of imaging vasospasms, but it is invasive, and it is proven to be associated with the risk of mild neurological deficit as well as ischemic insults [4].

As angiographic vasospasm is strongly associated with delayed cerebral ischemia (DCI) and clinical outcome, clinical trials in the last few decades focused on prevention of these angiographic spasms. Despite all efforts, no new pharmacological agents have shown to improve patient outcome. As such, it has become clear that our understanding of the pathophysiology of SAH is incomplete and we need to reevaluate our concepts on the complex pathophysiological process following SAH. Angiographic vasospasm is probably important.

The case fatality in aneurysmal subarachnoid hemorrhage (aSAH) is 50% due to the initial hemorrhage or subsequent complications like aneurysm rebleeding or delayed cerebral ischemia (DCI).

One factor that might influence the initial brain damage or subsequent complications is the use of antiplatelet medication before the initial hemorrhage.

Improvements in multidisciciplinary neurocritical care and advancements in medical and surgical treatment have contributed to a decline in the case fatality rate of aneurysmal subarachnoid hemorrhage [5].

A greater proportion of patients, therefore, are surviving their initial hemorrhagic event but remain at increased risk of a number of complications.

The case fatality after aneurysmal haemorrhage is 50%; one in eight patients with subarachnoid haemorrhage dies outside hospital. Rebleeding is the most imminent danger; a first aim is therefore occlusion of the aneurysm [6].

Prothrombotic states of early brain injury (EBI) and delayed cerebral ischemia (DCI) after aSAH determine morbidity and mortality.

The outcome depends on their condition on arrival at the hospital. However, a small number of patients recover from an initially poor condition.

Associated with intracerebral hematoma (ICH) typically has a poor outcome. SAH with ICH tends to have a worse prognosis than SAH alone.

It has a high socioeconomic impact as it tends to affect younger patients. The NCEPOD study looking into the management of aSAH has recommended that neurovascular units in the United Kingdom should aim to secure cerebral aneurysms within 48 hours and that delays because of weekend admissions can increase the mortality and morbidity attributed to aSAH.

A study provides important data showing excess in-Hospital mortality of patients with SAH on weekend admissions served by the United Kingdom's National Health Service.; However, there were no effects of weekend admission on long-term outcomes [7].

see also Subarachnoid hemorrhage outcome.

Aneurysmal subarachnoid hemorrhage complications

Aneurysmal subarachnoid hemorrhage complications.

Quality of life

Quality of life after aneurysmal subarachnoid hemorrhage.

Prognosis in Elderly Patients

Aneurysmal Subarachnoid Hemorrhage Outcome in Elderly Patients.

Cognitive outcome

Cognitive outcome after aneurysmal subarachnoid hemorrhage.

Obesity

Obesity in aneurysmal subarachnoid hemorrhage.

Dehydration

Dehydration on Prognosis in Aneurysmal Subarachnoid Hemorrhage

Factors

CSF metabolomics

Several studies have investigated the changes in CSF metabolomics that occur after aSAH. These studies have identified alterations in various metabolites and metabolic pathways, including those involved in energy metabolism, amino acid metabolism, and lipid metabolism.

One study found that levels of lactate, a marker of anaerobic metabolism, were significantly increased in the CSF of aSAH patients compared to controls. This suggests that there is a shift towards anaerobic metabolism in the brain following aSAH, possibly due to decreased oxygen delivery and

increased metabolic demand.

Other studies have reported alterations in amino acid metabolism, particularly involving glutamate and gamma-aminobutyric acid (GABA). Glutamate is an excitatory neurotransmitter that can lead to neuronal damage when present in excess, while GABA is an inhibitory neurotransmitter that can protect against excitotoxicity. Studies have shown that CSF levels of glutamate are increased and GABA levels are decreased in aSAH patients, which may contribute to the pathophysiology of the disease.

Alterations in lipid metabolism have also been reported in aSAH patients, with decreased levels of sphingomyelins and phosphatidylcholines in the CSF. These lipids play important roles in cellular membrane structure and function, and their depletion may contribute to neuronal damage and inflammation.

Overall, the findings of metabolomics studies suggest that aSAH leads to widespread metabolic alterations in the brain, involving multiple metabolic pathways. These alterations may contribute to the pathophysiology of the disease and represent potential targets for therapeutic intervention.

NFE2L2

NFE2L2 SNP, rs10183914, is significantly associated with aneurysmal subarachnoid hemorrhage outcome. This is consistent with a clinically relevant pathophysiological role for oxidative and inflammatory brain injury due to blood and its breakdown products in aSAH. Furthermore, the findings support NRF2 as a potential therapeutic target following aSAH and other forms of intracranial hemorrhage [8)]

In a study by Hammer et al. from the Paracelsus Medical University, complications like pneumonia (β = 5.11; 95% CI = 1.75-8.46; p = 0.0031), sepsis (β = 9.54; 95% CI = 3.27-15.82; p = 0.0031), hydrocephalus (β = 4.63; 95% CI = 1.82-7.45; p = 0.0014), and delayed cerebral ischemia (DCI) (β = 3.38; 95% CI = 0.19-6.56; p = 0.038) were critical factors depending on the LOS in intensive care as well as decompressive craniectomy (β = 5.02; 95% CI = 1.35-8.70; p = 0.0077). All analyzed comorbidities such as hypertension, diabetes, hypothyroidism, cholesterolemia, and smoking history had no significant impact on the LOS in intensive care. LOS in intensive care (OR = 1.09; 95% CI = 1.03-1.15; p = 0.0023), as well as World Federation of Neurosurgical Societies grading for subarachnoid hemorrhage (OR = 3.72; 95% CI = 2.23-6.21; p < 0.0001) and age (OR = 1.06; 95% CI = 1.02-1.10; p = 0.0061), were significant factors that had an impact on the outcome after 1 year. Complications in intensive care but not comorbidities are associated with higher LOS in intensive care. LOS in intensive care is a modest but significant predictor of outcomes after subarachnoid hemorrhage [9)].

Prediction models

Aneurysmal subarachnoid hemorrhage outcome prediction models

Scales

National Institute of Health Stroke Scale

Barthel Index

Extended Glasgow Outcome Scale.

Modified Rankin Scale

In a tertiary care center in India, despite recent advances in the treatment of patients with aSAH, the morbidity and mortality rates have failed to improve significantly in unselected patients and natural cohorts. This may be attributed to the natural history of aSAH, and calls for new strategies to diagnose and treat such patients before the catastrophe [10].

In the series of Nieuwkamp et al., despite an increase in the mean age of patients with SAH, case-fatality rates have decreased by 17% between 1973 and 2002 and show potentially important regional differences. This decrease coincides with the introduction of improved management strategies [11].

The case fatality after aneurysmal haemorrhage is 50%; one in eight patients with subarachnoid haemorrhage dies outside hospital.

Mortality is 10% within first few days

30-day mortality rate was 46% in one series, and in others over half the patients died within 2 weeks of their SAH.

overall mortality is 45% (range: 32—67%)

causes of mortality

neurogenic stunned myocardium

about 8% die from progressive deterioration from the initial hemorrhage

of those reaching neurosurgical care, vasospasm kills 7%, and causes severe deficit in another 7%.

about 30% of survivors have moderate to severe disability.

about 66 % of those who hove successful aneurysm clipping never return to the same quality of life as before the SAH.

With the limitation of an explorative cohort study the results indicate that routine transcranial doppler (TCD) studies do not improve the overall outcome of patients after aSAH [12].

Neutrophil-to-albumin ratio

The prognosis of aneurysmal subarachnoid hemorrhage (aSAH) survivors is concerning.

The goal of Zhang et al. in a study was to investigate and demonstrate the relationship between the neutrophil-to-albumin ratio (NAR) and long-term mortality of aSAH survivors. A retrospective observational cohort study was conducted at Sichuan University West China Hospital between January 2009 and June 2019. The investigation of the relationship between NAR and long-term mortality was conducted using univariate and multivariable Cox regression models. To demonstrate the predictive performance of different biomarkers over time, time-dependent receiver operating characteristic curve (ROC) analysis and decision curve analysis (DCA) were created.

In total, 3173 aSAH patients were included in this study. There was a strong and continuous relationship between NAR levels and long-term mortality (HR 3.23 95% CI 2.75-3.79, $p < 0.001$). After adjustment, the result was still significant (adjusted HR 1.78 95% CI 1.49-2.12). Compared with patients with the lowest quartile (< 0.15) of NAR levels, the risk of long-term mortality in the other groups was higher (0.15-0.20: adjusted HR 1.30 95% CI 0.97-1.73; 0.20-0.28: adjusted HR 1.37 95% CI 1.03-1.82; >0.28: adjusted HR 1.74 95% CI 1.30-2.32). Results in survivors were found to be still robust. Moreover, out of all the inflammatory markers studied, NAR demonstrated the highest correlation with long-term mortality.

A high level of Neutrophil to albumin ratio was associated with increased long-term mortality among patients with aneurysmal subarachnoid hemorrhage. Neutrophil to albumin ratio was a promising inflammatory marker for the long-term mortality of aSAH [13].

Amount of Bleeding

Quantitative estimation of the hemorrhage volume associated with aneurysm rupture is a tool of assessing prognosis.

A prospective cohort of 206 patients consecutively admitted with the diagnosis of aneurysmal subarachnoid hemorrhage to Hospital 12 de Octubre were included in the study. Subarachnoid, intraventricular, intracerebral, and total bleeding volumes were calculated using analytic software. For assessing factors related to prognosis, univariate and multivariate analysis (logistic regression) were performed. The relative importance of factors in determining prognosis was established by calculating their proportion of explained variation. Maximum Youden index was calculated to determine the optimal cut point for subarachnoid and total bleeding volume.

Variables independently related to prognosis were clinical grade at admission, age, and the different bleeding volumes. The proportion of variance explained is higher for subarachnoid bleeding. The optimal cut point related to poor prognosis is a volume of 20 mL both for subarachnoid and total bleeding.

Volumetric measurement of subarachnoid or total bleeding volume are both independent prognostic factors in patients with aneurysmal subarachnoid hemorrhage. A volume of more than 20 mL of blood in the initial noncontrast computed tomography is related to a clear increase in poor outcome risk [14].

Acute respiratory distress syndrome

Acute lung injury or acute respiratory distress syndrome (ALI/ARDS) is a common complication after aneurysmal subarachnoid hemorrhage (aSAH), and is associated with worse neurologic outcomes and longer hospitalization. However, the effect of ALI/ARDS in SAH has not been well elucidated. The purpose of this study was to determine the incidence of ALI/ARDS in a cohort of patients with SAH and to determine the risk factors for ALI/ARDS and their impact on patient prognosis. We performed a retrospective analysis of 167 consecutive patients with aSAH enrolled. ALI/ARDS patients were rigorously adjudicated using North American-European Consensus Conference definition. Regression analyses were used to test the risk factors for ALI/ARDS in patients with SAH. A total of 167 patients fulfilled the inclusion criteria, and 27% patients (45 of 167) developed ALI. Among all 45 ALI patients, 33 (20%, 33 of 167) patients met criteria for ARDS. On multivariate analysis, elderly patients, lower glasgow coma scale (GCS), higher Hunt-Hess grade, higher simplified acute physiology score (SAPS) II score, pre-existing pneumonia, gastric aspiration, hypoxemia, and tachypnea were the strongest risk factor for ALI/ARDS. Patients with ALI/ARDS showed worse clinical outcomes measured at 30 days. Development of ALI/ARDS was associated with a statistically significant increasing the odds of tracheostomy and hospital complications, and increasing duration of mechanical ventilation, intensive care unit (ICU) length and hospitalization stay. Development of ALI/ARDS is a severe complication of SAH and is associated with a poor clinical outcome, and further studies should focus on both prevention and management strategies specific to SAH-associated ALI/ARDS [15].

C-reactive protein

see C-reactive protein for aneurysmal subarachnoid hemorrhage outcome.

IL-6

Higher early IL6 serum levels after aSAH are associated with poor outcome at discharge. In addition, involvement of leukemia inhibitory factor (LIF) in the early inflammatory reaction after aSAH has been demonstrated [16].

APOEε4 polymorphism

The APOEε4 polymorphism was analysed in 147 patients with aSAH. Allele and genotype frequencies were compared to those found in a gender- and area-matched control group of healthy individuals (n = 211). Early cerebral vasospasm (CVS) was identified and treated according to neurointensive care unit (NICU) guidelines. Neurological deficit(s) at admittance and at 1-year follow-up visit was recorded. Neurological outcome was assessed by the National Institute of Health Stroke Scale, Barthel Index and the Extended Glasgow Outcome Scale.

APOEε4 and non-APOEε4 allele frequencies were similar in aSAH patients and healthy individuals. The presence of APOEε4 was not associated with the development of early CVS. We could not find an influence of the APOE polymorphism on 1-year neurological outcome between groups. Subgroup analyses of patients treated with surgical clipping vs endovascular coiling did not reveal any associations.

For Csajbok et al. APOEε4 polymorphism has no major influence on risk of aSAH, the occurrence of CVS or long-term neurological outcome after aSAH [17].

For Cheng et al., Apolipoprotein E (APOEε4) may induce cerebral perfusion impairment in the early phase, contributing to early brain injury (EBI) following aneurysmal subarachnoid hemorrhage (aSAH), and assessment of APOE genotypes could serve as a useful tool in the prognostic evaluation and therapeutic management of aSAH [18].

Direct oral anticoagulants or vitamin K antagonists

Iatrogenic coagulopathy caused by Direct oral anticoagulants or vitamin K antagonists was not associated with more severe radiological or clinical subarachnoid hemorrhage or worse clinical outcomes in hospitalized SAH patients [19].

Brain edema in aneurysmal subarachnoid hemorrhage

Brain edema in aneurysmal subarachnoid hemorrhage

Myosteatosis was found to be associated with poor physical condition directly after the onset of aSAH. Skeletal muscle atrophy and myosteatosis were however irrelevant to outcome in the Western-European aSAH patient. Future studies are needed to validate these finding [20].

Length of stay

Aneurysmal subarachnoid hemorrhage length of stay.

Brain Tissue Oxygenation Monitoring

A low PbtO2 value is associated with a worse prognosis, and an increase in the PbtO2 value in response to treatment is a marker of a good outcome [21].

Blood pressure

The inverse correlation between mean arterial pressure and mean transit time (MTT) in early perfusion computed tomography, increasing with the severity of aSAH, suggests an increasing disturbance of cerebral autoregulation with the severity of early brain injury. The results emphasize

the importance of maintaining physiological blood pressure values in the early phase of aSAH and preventing hypotension, especially in patients with poor-grade aSAH [22].

Cumulative Radiation Exposure

Survivors of Aneurysmal Subarachnoid Hemorrhage are exposed to high levels of medical radiation. The eyes are particularly at risk, with most patients exposed to levels that induce lens damage. This highlights the importance of strategies to reduce incidental and cumulative medical radiation exposure in this population [23]

References

1)
Yang Y, Li J, Xiao Z, Yang X, Wang L, Duan Y, Zhao K, Liu A. Relationship between stress hyperglycemia ratio and prognosis in patients with aneurysmal subarachnoid hemorrhage: a two-center retrospective study. Neurosurg Rev. 2024 Jul 12;47(1):315. doi: 10.1007/s10143-024-02549-z. PMID: 38992256.
2)
Malhotra K, Conners JJ, Lee VH, Prabhakaran S. Relative changes in transcranial Doppler velocities are inferior to absolute thresh- olds in prediction of symptomatic vasospasm after subarachnoid hemorrhage. J Stroke Cerebrovasc Dis. 2014;23:31–6.
3)
Jabbarli R, Gläsker S, Weber J, Taschner C, Olschewski M, Velthoven VV. Predictors of severity of cerebral vasospasm caused by aneurysmal subarachnoid hemorrhage. J Stroke Cerebrovasc Dis. 2013;22:1332–9.
4)
Zhang H, Zhang B, Li S, Liang C, Xu K, Li S. Whole brain CT perfusion combined with CT angiography in patients with sub- arachnoid haemorrhage and cerebral vasospasm. Clin Neurol Neurosurg. 2013;115:2496–501.
5)
Mackey J, Khoury JC, Alwell K, Moomaw CJ, Kissela BM, Flaherty ML, et al: Stable incidence but declining case-fatali- ty rates of subarachnoid hemorrhage in a population. Neurol- ogy 87:2192–2197, 2016
6)
van Gijn J, Kerr RS, Rinkel GJ. Subarachnoid haemorrhage. Lancet. 2007 Jan 27;369(9558):306-18. Review. PubMed PMID: 17258671.
7)
Deshmukh H, Hinkley M, Dulhanty L, Patel HC, Galea JP. Effect of weekend admission on in-Hospital mortality and functional outcomes for patients with acute subarachnoid haemorrhage (SAH). Acta Neurochir (Wien). 2016 May;158(5):829-35. doi: 10.1007/s00701-016-2746-z. Epub 2016 Mar 1. PubMed PMID: 26928730; PubMed Central PMCID: PMC4826657.
8)
Gaastra B, Duncan P, Bakker MK, Hostettler IC, Alg VS, Houlden H, Ruigrok YM, Galea I, Tapper W, Werring D, Bulters D. Genetic variation in NFE2L2 is associated with outcome following aneurysmal subarachnoid haemorrhage. Eur J Neurol. 2022 Sep 23. doi: 10.1111/ene.15571. Epub ahead of print. PMID: 36148820.
9)
Hammer A, Ranaie G, Erbguth F, Hohenhaus M, Wenzl M, Killer-Oberpfalzer M, Steiner HH, Janssen H. Impact of Complications and Comorbidities on the Intensive Care Length of Stay after Aneurysmal Subarachnoid Haemorrhage. Sci Rep. 2020 Apr 10;10(1):6228. doi: 10.1038/s41598-020-63298-9.

PubMed PMID: 32277142.
10)

Sodhi HB, Savardekar AR, Mohindra S, Chhabra R, Gupta V, Gupta SK. The clinical profile, management, and overall outcome of aneurysmal subarachnoid hemorrhage at the neurosurgical unit of a tertiary care center in India. J Neurosci Rural Pract. 2014 Apr;5(2):118-26. doi: 10.4103/0976-3147.131650. PubMed PMID: 24966547.
11)

Nieuwkamp DJ, Setz LE, Algra A, Linn FH, de Rooij NK, Rinkel GJ. Changes in case fatality of aneurysmal subarachnoid haemorrhage over time, according to age, sex, and region: a meta-analysis. Lancet Neurol. 2009 Jul;8(7):635-42. doi: 10.1016/S1474-4422(09)70126-7. Epub 2009 Jun 6. PubMed PMID: 19501022.
12)

Ehrlich G, Kirschning T, Wenz H, Hegewald AA, Groden C, Schmiedek P, Scharf J, Seiz-Rosenhagen M. Is there an influence of routine daily transcranial doppler examination on clinical outcome in patients after aneurysmal subarachnoid hemorrhage? World Neurosurg. 2016 Jan 5. pii: S1878-8750(15)01714-3. doi: 10.1016/j.wneu.2015.11.091. [Epub ahead of print] PubMed PMID: 26768855.
13)

Zhang R, Zhang Y, Liu Z, Pei Y, He Y, Yu J, You C, Ma L, Fang F. Association between neutrophil-to-albumin ratio and long-term mortality of aneurysmal subarachnoid hemorrhage. BMC Neurol. 2023 Oct 19;23(1):374. doi: 10.1186/s12883-023-03433-x. PMID: 37858065.
14)

Lagares A, Jiménez-Roldán L, Gomez PA, Munarriz PM, Castaño-León AM, Cepeda S, Alén JF. Prognostic Value of the Amount of Bleeding After Aneurysmal Subarachnoid Hemorrhage: A Quantitative Volumetric Study. Neurosurgery. 2015 Dec;77(6):898-907. doi: 10.1227/NEU.0000000000000927. PubMed PMID: 26308629.
15)

Wu J, Gao W, Zhang H. Development of acute lung injury or acute respiratory distress syndrome after subarachnoid hemorrhage, predictive factors, and impact on prognosis. Acta Neurol Belg. 2023 Mar 15. doi: 10.1007/s13760-023-02207-z. Epub ahead of print. PMID: 36922484.
16)

Höllig A, Remmel D, Stoffel-Wagner B, Schubert GA, Coburn M, Clusmann H. Association of early inflammatory parameters after subarachnoid hemorrhage with functional outcome: A prospective cohort study. Clin Neurol Neurosurg. 2015 Aug 28;138:177-183. doi: 10.1016/j.clineuro.2015.08.030. [Epub ahead of print] PubMed PMID: 26355810.
17)

Csajbok LZ, Nylén K, Öst M, Blennow K, Zetterberg H, Nellgård P, Nellgård B. Apolipoprotein E polymorphism in aneurysmal subarachnoid haemorrhage in West Sweden. Acta Neurol Scand. 2015 Sep 16. doi: 10.1111/ane.12487. [Epub ahead of print] PubMed PMID: 26374096.
18)

Cheng C, Jiang L, Yang Y, Wu H, Huang Z, Sun X. Effect of APOE Gene Polymorphism on Early Cerebral Perfusion After Aneurysmal Subarachnoid Hemorrhage. Transl Stroke Res. 2015 Sep 14. [Epub ahead of print] PubMed PMID: 26370543.
19)

Veldeman M, Rossmann T, Weiss M, Conzen-Dilger C, Korja M, Hoellig A, Virta JJ, Satopää J, Luostarinen T, Clusmann H, Niemelä M, Raj R. Aneurysmal Subarachnoid Hemorrhage in Hospitalized Patients on Anticoagulants-A Two Center Matched Case-Control Study. J Clin Med. 2023 Feb 13;12(4):1476. doi: 10.3390/jcm12041476. PMID: 36836011; PMCID: PMC9958876.
20)

Shen Y, Levolger S, Zaid Al-Kaylani AHA, Uyttenboogaart M, van Donkelaar CE, Van Dijk JMC, Viddeleer AR, Bokkers RPH. Skeletal muscle atrophy and myosteatosis are not related to long-term aneurysmal subarachnoid hemorrhage outcome. PLoS One. 2022 Mar 4;17(3):e0264616. doi:

10.1371/journal.pone.0264616. PMID: 35245308.

21)

Gouvea Bogossian E, Battaglini D, Fratino S, Minini A, Gianni G, Fiore M, Robba C, Taccone FS. The Role of Brain Tissue Oxygenation Monitoring in the Management of Subarachnoid Hemorrhage: A Scoping Review. Neurocrit Care. 2023 Feb 17. doi: 10.1007/s12028-023-01680-x. Epub ahead of print. PMID: 36802011.

22)

Hofmann BB, Donaldson DM, Fischer I, Karadag C, Neyazi M, Piedade GS, Abusabha Y, Muhammad S, Rubbert C, Hänggi D, Beseoglu K. Blood Pressure Affects the Early CT Perfusion Imaging in Patients with aSAH Reflecting Early Disturbed Autoregulation. Neurocrit Care. 2023 Feb 17. doi: 10.1007/s12028-023-01683-8. Epub ahead of print. PMID: 36802010.

23)

Asundi SH, Plummer MP, Sundararajan K, O'Callaghan G, Kar P, Jukes A, Boyd CM, Chen W, Dong C, Webber T. Cumulative Radiation Exposure Post Aneurysmal Subarachnoid Hemorrhage. Clin Neuroradiol. 2025 Mar 31. doi: 10.1007/s00062-025-01513-8. Epub ahead of print. PMID: 40164929.