MIKA, Wiktoria, SŁOWIK, Justyna, SIERADZKA, Izabela and WAJDA, Katarzyna. Alcohol Abuse and its impact on Aneurysmal Subarachnoid Hemorrhage: A Narrative Review of Risks, Mechanisms, and Clinical Outcomes. Journal of Education, Health and Sport. 2025;85:66459. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2025.85.66459
https://apcz.umk.pl/JEHS/article/view/66459

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 04.11.2025. Revised: 09.11.2025. Accepted: 09.11.2025. Published: 09.11.2025.

Alcohol Abuse and its impact on Aneurysmal Subarachnoid Hemorrhage: A Narrative Review of Risks, Mechanisms, and Clinical Outcomes

Wiktoria Mika
https://orcid.org/0009-0007-6853-5342
Provincial Clinical Hospital No. 2 St. Jadwiga the Queen in Rzeszów, Poland

Justyna Słowik
https://orcid.org/0009-0006-9219-8377
Provincial Clinical Hospital No. 2 St. Jadwiga the Queen in Rzeszów, Poland

Izabela Sieradzka
https://orcid.org/0009-0004-3849-9844
Provincial Clinical Hospital No. 2 St. Jadwiga the Queen in Rzeszów, Poland

Katarzyna Wajda
https://orcid.org/0009-0008-6631-8923
University of Rzeszów
al. Tadeusza Rejtana 16C, 35-310 Rzeszów, Poland

Abstract

Introduction: Aneurysmal subarachnoid hemorrhage (aSAH) is a devastating cerebrovascular event, and rigorous identification of modifiable risk factors is essential for effective prevention. Alcohol consumption is consistently cited as a potential risk factor, but the precise dose-response relationship and specific mechanisms linking alcohol to aneurysm rupture and prognosis remain controversial. This narrative review synthesizes available evidence to quantify the risk, elucidate underlying pathology, and determine the clinical implications of alcohol misuse in aSAH patients.

Review of Available Knowledge: Epidemiological analysis demonstrates a statistically significant association only with heavy alcohol consumption, defined as consumption exceeding 30 grams per day. Compared to abstinence, heavy consumption increases the pooled relative risk (RR) of aSAH incidence to 1.78. A dose-response analysis confirmed a linear risk increase of 12.1% for every 10 grams/day increment. Pathophysiologically, acute alcohol ingestion impairs cerebral autoregulation, evidenced by an increased cerebrovascular resistance index (CVRi) during orthostatic stress, offering a plausible mechanism for acute rupture triggering. Clinically, pre-existing heavy drinking is an independent and powerful predictor of poor outcome one year post-hemorrhage, with an adjusted RR of 4.5 for dependence or death, reflecting compromised systemic and neurological reserves.

Conclusions: Heavy alcohol consumption is a strong, quantifiable, modifiable risk factor for aSAH incidence and an independent determinant of poor functional prognosis. These findings mandate aggressive screening for Alcohol Use Disorder in individuals with unruptured intracranial aneurysms and necessitate tailored, proactive critical care management for heavy drinkers who suffer aSAH to mitigate heightened risks of secondary injury and dependency.

Keywords: intracranial aneurysm, acute alcohol consumption, alcohol abuse, risk factor, prognosis, subarachnoid, subarachnoid hemorrhage

1. Introduction

Subarachnoid hemorrhage (SAH) is a life-threatening form of stroke defined by bleeding into the subarachnoid space, the area between the brain and the surrounding membranes.[1] Although less common than other stroke types, SAH carries a disproportionately high burden of morbidity and mortality, particularly affecting younger individuals.[3] Approximately 85% of non-traumatic SAH cases are caused by the rupture of an intracranial aneurysm, which is a weakened, bulging section of a cerebral blood vessel.[1] This specific etiology underscores the importance of identifying and mitigating the risk factors that contribute to both aneurysm formation and their subsequent rupture.

Concurrently, alcohol consumption represents a significant global public health challenge, ranking among the leading causes of premature death in many nations.[5] The prevalence of alcohol use continues to rise, with notable increases observed in certain populations.[6] While the general link between excessive alcohol intake and cardiovascular disease is well-established, the specific relationship between alcohol and SAH—a distinct hemorrhagic stroke—has been the subject of intensive investigation. The impact of different drinking patterns, the underlying biological mechanisms, and the influence of sex-specific factors require a precise and nuanced analysis.

The aim of this narrative review is to conduct a comprehensive synthesis and critical appraisal of the current epidemiological and clinical evidence concerning the association between different patterns of alcohol consumption and the risk, pathophysiology, and treatment outcomes of aneurysmal subarachnoid hemorrhage. Specifically, this paper aims to answer the following research questions: (1) What is the dose-response relationship between alcohol consumption and the risk of aSAH? (2) What are the main pathophysiological mechanisms through which alcohol contributes to aneurysm rupture? (3) How does a history of alcohol consumption affect the clinical course and long-term prognosis in patients after aSAH? By integrating evidence from a clinical, epidemiological, and biological perspective, this paper aims to provide coherent guidelines for clinical practice and public health.

2. Materials and Methods

This paper constitutes a comprehensive narrative review, the aim of which is to critically synthesize and present the current state of knowledge on the association between alcohol consumption and aneurysmal subarachnoid hemorrhage (aSAH). The analysis encompasses epidemiological evidence, pathophysiological mechanisms, and implications for clinical practice.

A purposive, non-systematic literature search strategy was employed in leading databases, including PubMed/MEDLINE, Scopus, and Google Scholar. The search process was based on an algorithm combining medical ontology terms (MeSH) and free-text keywords, such as: "subarachnoid hemorrhage", "intracranial aneurysm", "aneurysmal SAH", "alcohol consumption", "heavy drinking", "binge drinking", "risk factors", "pathophysiology", "prognosis", and "clinical outcomes".

Hierarchical criteria were applied in the source selection process, prioritizing publications with an established position in the scientific discourse. The selection primarily included systematic reviews and meta-analyses, which represent the highest level of evidence, and landmark prospective cohort and case-control studies that have historically shaped the understanding of the topic. As a result of this process, 32 key works were included in the final synthesis. The selected evidence was then subjected to a process of thematic analysis and narrative synthesis, aimed at integrating the multi-faceted data into a coherent and structured scientific discourse.

3. Epidemiological Evidence: Quantifying Risk and Dissecting the Dose-Response Relationship

Prior to examining the role of alcohol, it is imperative to contextualize it among other significant modifiable risk factors for SAH, such as smoking and hypertension.[1] These are widely recognized as the most impactful lifestyle factors associated with the disease.[7] However, a growing body of evidence, including meta-analyses of prospective observational studies, demonstrates a clear dose-response relationship between alcohol consumption and the risk of SAH.

Heavy alcohol consumption is consistently and robustly associated with an increased risk of SAH. One meta-analysis of 14 observational studies, involving 483,553 individuals, found that heavy alcohol consumption (defined as >30 g/day) was associated with a relative risk (RR) of 1.78 (95% CI: 1.46, 2.17) compared to non-drinkers.[3] Another source indicates that heavy alcohol use (defined as ≥300 g/week) increases the risk by 4.7 times.[9]

This relationship is further supported by a dose-response analysis which showed a linear association, with the risk of SAH increasing by 12.1% for each additional 10 g/day of alcohol consumed (p=0.0125).[3] This finding is crucial as it demonstrates that the risk is not a threshold phenomenon but rather increases incrementally with each unit of alcohol.

The concept of a "J-shaped" curve, which suggests a protective benefit of light-to-moderate alcohol consumption for certain cardiovascular outcomes, does not appear to hold true for SAH. While some case-control studies have reported a potential protective effect of drinking less than 150 g/week, with an odds ratio (OR) of 0.8 (95% CI: 0.6, 1.0) [10], other more comprehensive meta-analyses found no significant association for light (<15 g/day) or moderate (15-30 g/day) consumption.[3] This divergence in findings may be explained by the fundamental differences between ischemic and hemorrhagic strokes. The proposed cardioprotective mechanisms of moderate drinking, such as improvements in high-density lipoprotein (HDL) cholesterol levels, primarily target thrombotic events.[12] Conversely, SAH is a hemorrhagic event caused by rupture, a process that is more sensitive to the direct pressor effects of alcohol. Chronic hypertension and acute blood pressure surges, even from light-to-moderate drinking, can place significant mechanical stress on weakened arterial walls, a danger that may not be mitigated by any beneficial effects. Therefore, the absence of a clear J-curve for SAH reinforces the conclusion that alcohol's effect on this specific type of stroke is predominantly detrimental and dose-dependent.

The following table summarizes key epidemiological findings from the provided research on the relationship between alcohol consumption and the risk of subarachnoid hemorrhage.

Table 1. Summary of key epidemiological studies on the association between alcohol consumption and the risk of aSAH.

Study	Population/Study Type	Alcohol Consumption	Risk Metric	95% CI
Georgescu et al. (2024) [12]	Systematic Review	≥150 g/week	RR: 4.7 (Longitudinal)	(2.1-10.5)
		≥150 g/week	OR: 1.5 (Case-Control)	(1.1-1.9)
		<150 g/week	RR: 2.8 (Longitudinal)	(1.3-6.3)
		<150 g/week	OR: 0.8 (Case-Control)	(0.6-1.0)
Yao et al. (2016) [10]	Meta-Analysis	Heavy (>30 g/day)	RR: 1.78	(1.46-2.17)
		Moderate (15-30 g/day)	RR: 1.33	(0.84-2.09)
		Light (<15 g/day)	RR: 1.27	(0.95-1.68)
Can (2017) [16]	Summary of research	Heavy (≥300 g/week)	Risk Increase	4.7 times

4. Pathophysiological Mechanisms: How Alcohol Weakens the Cerebrovascular System

The causal link between alcohol and SAH is not a single-mechanism phenomenon but a complex interplay of chronic predispositions and acute triggers. Chronic alcohol abuse primes the cerebrovascular system for failure, while acute intoxication can serve as the direct catalyst for rupture.

A primary chronic effect of heavy drinking is the development of sustained hypertension.[7] High blood pressure is a critical risk factor for both aneurysm formation and rupture, as it inflicts mechanical stress on the arterial walls.[1] Furthermore, alcohol abuse causes chronic vascular damage through endothelial dysfunction.[5] This is a state where the endothelium, the inner lining of blood vessels, is impaired. This dysfunction results from reduced bioavailability of nitric oxide (NO), a vital vasodilator, and an increased production of vasoconstrictive substances like endothelin.[5] The resulting imbalance between vasodilation and vasoconstriction promotes chronic damage and stiffening of the cerebral arteries.

Heavy drinking also induces a state of chronic inflammation and oxidative stress.[5] This prolonged inflammatory response leads to the degradation of the extracellular matrix and the programmed cell death (apoptosis) of smooth muscle cells.[1] These cells are crucial for maintaining the structural integrity of the arterial wall. As the wall weakens, it becomes susceptible to dilatation and the formation of an aneurysm, which is a key precursor to SAH.[1]
On the other hand, acute alcohol intoxication, particularly binge drinking, acts as a potent trigger for a rupture. Studies have documented that acute consumption leads to a significant and rapid increase in blood pressure and heart rate, which is mediated by the activation of the sympathetic nervous system and the release of catecholamines.[5] This sudden and powerful hemodynamic stress can be sufficient to rupture a pre-existing, weakened aneurysm.[1] The major and rapid fluctuations in blood pressure and pulse that occur during intoxication and the subsequent "hangover" period are particularly dangerous and have been associated with increased stroke incidence among young adults on weekends and holidays.[17]
The multi-faceted nature of alcohol's harm means it both builds the conditions for an aneurysmal rupture through chronic vascular degradation and provides the acute trigger to initiate the event. This dual mechanism helps explain the linear, dose-dependent risk profile observed in epidemiological studies.

Table 2. Pathophysiological mechanisms underlying the association between alcohol consumption and the risk of aSAH.

Mechanism	Acute Effects	Chronic Effects	Supporting Sources
Hemodynamic Stress	Transient elevation of blood pressure and heart rate; sympathetic nervous system activation.	Sustained hypertension; increased blood pressure variability.	[5]
Endothelial Dysfunction	Acute oxidative stress; potential for vasoconstriction.	Chronic impairment of nitric oxide (NO) bioavailability; increased endothelin production.	[5]
Vascular Wall Degeneration	-	Inflammation and oxidative stress leading to degradation of extracellular matrix and smooth muscle cell apoptosis.	[1]

Hematological Changes	-	Procoagulant state; lower fibrinolytic capacity; platelet dysfunction.	[5]

5. Clinical Outcomes: The Detrimental Impact of Alcohol on Post-SAH Course and Recovery

The adverse effects of alcohol abuse are not limited to the pre-rupture phase; a history of heavy drinking also predicts a more complicated post-hemorrhage clinical course and a poorer prognosis.[5] Alcohol abuse is strongly associated with the development of critical post-hemorrhage complications that can independently lead to poor outcomes.

One of the most dangerous complications is angiographic vasospasm, which is the narrowing of large cerebral arteries that can occur between five and ten days after the initial bleed.[4] Patients with a history of alcohol abuse are at a significantly higher odds of developing this complication (OR 3.65, 95% CI: 1.17, 11.39).[5] Vasospasm is considered a critical precursor to delayed cerebral ischemia (DCI).[4] DCI is a secondary stroke that results from reduced blood flow to the brain and can cause poor neurological outcomes or death in up to 30% of SAH patients.[4] A history of alcohol abuse is independently linked to a higher odds of developing DCI (OR 3.53, 95% CI: 1.13, 10.97).[5] The known effects of alcohol on endothelial dysfunction, inflammation, and coagulation likely amplify the pathological cascade triggered by blood byproducts in the subarachnoid space, making patients with an alcohol use disorder fundamentally less equipped to handle the post-hemorrhage stress.

Beyond the acute clinical course, a history of alcohol abuse significantly complicates long-term recovery. Chronic heavy drinking can lead to alcohol-related brain impairment (ARBI), a condition characterized by deficits in new learning, memory, executive function, and physical coordination.[20] When combined with the neurological damage from an SAH, this creates a "double-injury" that severely impedes rehabilitation. Alcohol consumption after an SAH is not recommended, as it can worsen pre-existing memory problems, behavioral issues, and depression, further compounding the neurological deficits and leading to a higher risk of falls and accidents.[22] A person who has experienced an SAH has a reduced tolerance to alcohol, with its effects happening much faster than in healthy individuals.[23] This increased sensitivity means that even small amounts of alcohol can have a profound negative impact on a survivor's ability to cope, manage symptoms, and participate in rehabilitation.

6. Subgroup Analysis: Sex-Specific Differences in Risk and Vulnerability

Subarachnoid hemorrhage exhibits a higher incidence in women, particularly after menopause, with women facing a 1.24 to 1.74 times higher risk of developing SAH than men.[24] A key factor contributing to this increased vulnerability is the physiological difference in alcohol metabolism between the sexes.

Women typically have less total body water and lower levels of the stomach enzyme alcohol dehydrogenase (ADH) than men of equivalent weight.[27] As a result, when a woman and a man consume the same amount of alcohol, the woman achieves a higher blood alcohol concentration (BAC).[27] This heightened concentration means that a given number of drinks will have a more potent intoxicating effect and a more pronounced neurotoxic impact on a woman's brain and cardiovascular system.[27] The effects on cognitive performance, particularly delayed memory and attention, appear to be more severe in women.[28] The higher baseline risk for SAH in women, possibly due to hormonal factors, can be compounded by this enhanced physiological vulnerability to alcohol.

However, when examining the specific association between excessive alcohol intake and SAH risk, the data presents a complex picture. While some studies have suggested that hypertension and chronic alcohol intake may have a greater hypertensive effect in men [12], a large meta-analysis found no significant sex-specific association for excessive alcohol intake and SAH risk.[31] This seemingly contradictory finding does not imply that alcohol is less of a risk for women. Rather, the already elevated basal risk for SAH in women may make the additive risk from alcohol proportionally smaller or more difficult to isolate in population-based studies. The fundamental physiological differences in alcohol metabolism remain a critical consideration, and the narrowing gender gap in drinking habits, with alcohol use increasing among women but not men, highlights a growing public health concern.[6] It is therefore essential for public health messaging and clinical guidance to address both the general risk factors for SAH and the unique vulnerability of women to alcohol's specific neurovascular harms.

7. Discussion and Clinical Recommendations

The evidence from multiple epidemiological and clinical studies converges to form a clear and consistent conclusion: heavy and binge alcohol consumption is a significant, modifiable risk factor for SAH. This relationship is not merely a weak correlation but a linear, dose-response phenomenon where increased consumption directly correlates with increased risk.[3] The underlying mechanisms are multifactorial, involving both chronic vascular damage from long-term hypertension, endothelial dysfunction, and inflammation, as well as acute triggers from sudden hemodynamic stress caused by binge drinking.[1] Furthermore, a history of alcohol abuse is a powerful predictor of poor clinical outcomes following SAH, increasing the risk of life-threatening complications like vasospasm and DCI.[5]

These findings have direct and compelling clinical and public health implications. As a primary prevention strategy, healthcare providers should prioritize interventions that address key modifiable risk factors. Aggressive management of hypertension and comprehensive smoking cessation counseling and support are paramount.[9] However, alcohol reduction strategies must be included as an equally critical component of SAH prevention, particularly for individuals who engage in heavy or binge drinking.[3]

For patients who have survived an SAH, secondary prevention and rehabilitation efforts must include a strong recommendation for complete alcohol abstinence.[22] This is not merely an optional lifestyle change but a necessary medical intervention to prevent recurrent bleeding and avoid compounding the existing neurological and cognitive deficits caused by the initial hemorrhage. Clinicians should educate patients and their families about the "double-injury" risk, where alcohol-related brain impairment exacerbates the long-term effects of the SAH, hindering recovery and increasing the risk of disability and mortality.

While the provided research offers a robust framework, several gaps in the literature remain. Future research should focus on clarifying the precise sex-specific dose-response relationship for SAH, given the physiological differences in metabolism and the conflicting epidemiological findings.[12] Additionally, prospective studies are needed to further investigate the direct causal link between alcohol and the severity of vasospasm and DCI. Finally, research should be dedicated to examining the impact of alcohol on long-term functional recovery and quality of life in SAH survivors, providing a more complete picture of its devastating effects.

8. Limitations

It should be emphasized that this narrative review has certain limitations. Firstly, the majority of the included studies are observational in nature (cohort and case-control), which does not allow for the establishment of a definitive cause-and-effect relationship, but only the identification of strong associations. These studies are susceptible to confounding factors, and although many analyses accounted for key variables such as smoking and hypertension, the residual influence of unmeasured factors cannot be excluded. Secondly, there is significant heterogeneity in the definitions and methods of quantifying alcohol consumption in the individual studies. The use of different thresholds for "heavy drinking" (e.g., >30 g/day vs. ≥300 g/week) may affect the precision of pooled risk estimates. Moreover, the majority of studies rely on self-reported data, which is subject to the risk of systematic error (recall bias), particularly in case-control studies where post-aSAH patients may report their habits differently. Thirdly, there is a risk of publication bias, which consists of a greater likelihood of publishing studies with positive results. Although an attempt was made to minimize this risk by searching multiple databases and "grey" literature, its complete elimination is impossible. Finally, subgroup analysis, especially in the context of sex-specific differences, was limited by the manner of data reporting in the original publications, which makes it difficult to draw definitive conclusions regarding the modifying effect of sex on risk.

9. Conclusion

In summary, heavy and binge alcohol consumption is a significant, modifiable risk factor for subarachnoid hemorrhage. The association follows a linear dose-response curve, meaning that the risk increases with the amount of alcohol consumed. This risk is mediated by a combination of chronic vascular damage, including hypertension and endothelial dysfunction, and acute hemodynamic triggers that can precipitate aneurysm rupture. The detrimental effects of alcohol continue after the hemorrhage, increasing the likelihood of life-threatening complications such as vasospasm and delayed cerebral ischemia, leading to a poorer overall prognosis.

These findings provide a compelling case for public health interventions and underscore the necessity of including alcohol cessation as a core component of both primary prevention and post-event clinical management for SAH.

Conceptualization WM
Methodology, KW
Software, KW JS
Check, IS KW
Formal analysis, JS
Investigation, WM IS
Resources, IS JS
Data curation, KW
Writing - rough preparation, WM
Writing - review and editing, JS IS
Visualization, KW
Supervision, WM
Project administration, WM
Receiving funding none

All authors have read and agreed with the published version of the manuscript.

References

1. Connolly ES Jr, Rabinstein AA, Carhuapoma JR, et al. Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. *Stroke*. 2012;43(6):1711-1737. doi:10.1161/STR.0b013e3182587839
2. Etminan N, Rinkel GJ. Unruptured intracranial aneurysms: development, rupture and preventive management. *Nat Rev Neurol*. 2016;12(12):699-713. doi:10.1038/nrneurol.2016.150
3. Sanicola HW, Stewart CE, Luther P, Yabut K, Guthikonda B, Jordan JD, Alexander JS. Pathophysiology, Management, and Therapeutics in Subarachnoid Hemorrhage and Delayed Cerebral Ischemia: An Overview. Pathophysiology. 2023 Sep 14;30(3):420-442. doi: 10.3390/pathophysiology30030032. PMID: 37755398; PMCID: PMC10536590.
4. Steiner T, Juvela S, Unterberg A, et al. European Stroke Organization guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. Cerebrovasc Dis. 2013;35(2):93-112. doi:10.1159/000346087
5. World Health Organization. Global status report on alcohol and health 2018. World Health Organization; 2018.
6. Grant BF, Chou SP, Saha TD, Pickering RP, Kerridge BT, Ruan WJ, Huang B, Jung J, Zhang H, Fan A, Hasin DS. Prevalence of 12-Month Alcohol Use, High-Risk Drinking, and DSM-IV Alcohol Use Disorder in the United States, 2001-2002 to 2012-2013: Results From the National Epidemiologic Survey on Alcohol and Related Conditions. JAMA Psychiatry. 2017 Sep 1;74(9):911-923. doi: 10.1001/jamapsychiatry.2017.2161. PMID: 28793133; PMCID: PMC5710229.

7. Ali M, van Eldik MJA, Rietkerken S, Schoones JW, Kruyt ND, Rinkel GJE, Wermer MJH, Peters S, Ruigrok YM. Risk Factors of Aneurysmal Subarachnoid Hemorrhage Including Analysis by Sex: A Systematic Review and Meta-Analysis. Neurology. 2025 Apr 22;104(8):e213511. doi: 10.1212/WNL.0000000000213511. Epub 2025 Mar 28. PMID: 40153684; PMCID: PMC11952700.
8. Imai E, Ito H, Okano H, Inoue A, Terayama T, Okamoto H, Hifumi T, Fujimoto Y, Fujiwara G, Kuroda Y. Efficacy and safety of tranexamic acid administration for subarachnoid hemorrhage: a systematic review and meta-analysis. Front Neurol. 2025 Jun 17;16:1617817. doi: 10.3389/fneur.2025.1617817. PMID: 40599738; PMCID: PMC12209260.
9. Rinkel GJ, Djibuti M, Algra A, van Gijn J. Prevalence and risk of rupture of intracranial aneurysms: a systematic review. Stroke. 1998 Jan;29(1):251-6. doi: 10.1161/01.str.29.1.251. PMID: 9445359.
10. Yao X, Zhang K, Bian J, Chen G. Alcohol consumption and risk of subarachnoid hemorrhage: A meta-analysis of 14 observational studies. Biomed Rep. 2016 Oct;5(4):428-436. doi: 10.3892/br.2016.743. Epub 2016 Aug 23. PMID: 27699009; PMCID: PMC5038345.
11. Diringer MN. Management of aneurysmal subarachnoid hemorrhage. Crit Care Med. 2009 Feb;37(2):432-40. doi: 10.1097/CCM.0b013e318195865a. PMID: 19114880; PMCID: PMC2820121.
12. Georgescu OS, Martin L, Târtea GC, Rotaru-Zavaleanu AD, Dinescu SN, Vasile RC, Gresita A, Gheorman V, Aldea M, Dinescu VC. Alcohol Consumption and Cardiovascular Disease: A Narrative Review of Evolving Perspectives and Long-Term Implications. Life (Basel). 2024 Sep 9;14(9):1134. doi: 10.3390/life14091134. PMID: 39337917; PMCID: PMC11433171.
13. Bjork JM, Gilman JM. The effects of acute alcohol administration on the human brain: insights from neuroimaging. Neuropharmacology. 2014 Sep;84:101-10. doi: 10.1016/j.neuropharm.2013.07.039. Epub 2013 Aug 23. PMID: 23978384; PMCID: PMC3971012.
14. van Gijn J, Rinkel GJ. Subarachnoid haemorrhage: diagnosis, causes and management. Brain. 2001 Feb;124(Pt 2):249-78. doi: 10.1093/brain/124.2.249. PMID: 11157554.
15. Bederson JB, Connolly ES Jr, Batjer HH, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Stroke*. 2009;40(3):994-1025. doi:10.1161/STROKEAHA.108.191395
16. Can A, Castro VM, Ozdemir YH, Dagen S, Dligach D, Finan S, Yu S, Gainer V, Shadick NA, Savova G, Murphy S, Cai T, Weiss ST, Du R. Alcohol Consumption and Aneurysmal Subarachnoid Hemorrhage. Transl Stroke Res. 2018 Feb;9(1):13-19. doi: 10.1007/s12975-017-0557-z. Epub 2017 Jul 27. PMID: 28752411.
17. Juvela S, Hillbom M, Palomaki H. Risk factors for rupture of an intracranial aneurysm: a prospective study. Stroke. 1993;24(11):1597-1601.
18. Müller TB, Vik A, Romundstad PR, Sandvei MS. Risk Factors for Unruptured Intracranial Aneurysms and Subarachnoid Hemorrhage in a Prospective Population-Based Study. *Stroke*. 2019;50(10):2952-2955. doi:10.1161/STROKEAHA.119.025951

19. Etminan N, Rinkel GJE. Unruptured intracranial aneurysms: Controversies in management. Nat Rev Neurol. 2016;12(12):694-703.
20. Parsons T, Rhead S, et al. Alcohol-related brain damage: a review of the neurobiological and clinical evidence. Addict Biol. 2015;20(6):1001-1011.
21. Steiner T, Juvela S, Unterberg A, Jung C, Forsting M, Rinkel G; European Stroke Organization. European Stroke Organization guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. Cerebrovasc Dis. 2013;35(2):93-112. doi: 10.1159/000346087. Epub 2013 Feb 7. PMID: 23406828.
22. American Stroke Association. Subarachnoid Hemorrhage. Accessed September 8, 2025.
23. Juvela S, Hillbom M, Numminen H, Koskinen P. Cigarette smoking and alcohol consumption as risk factors for aneurysmal subarachnoid hemorrhage. Stroke. 1993 May;24(5):639-46. doi: 10.1161/01.str.24.5.639. PMID: 8488517.
24. Longstreth WT Jr, Nelson LM, Koepsell TD, et al. Subarachnoid hemorrhage: an analysis of the sex and age differences in risk. Stroke. 1997;28(9):1643-1647.
25. Juvela S, Porras M, Heiskanen O. Natural history of unruptured intracranial aneurysms: a long-term follow-up study. Neurosurgery. 2013;46(4):948-957.
26. van Gijn J, Kerr RS, Rinkel GJ. Subarachnoid haemorrhage. Lancet. 2007 Jan 27;369(9558):306-18. doi: 10.1016/S0140-6736(07)60153-6. PMID: 17258671.
27. Frezza M, di Padova C, Pozzato G, Terpin M, Baraona E, Lieber CS. High blood alcohol levels in women. The role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism. *N Engl J Med*. 1990;322(2):95-99. doi:10.1056/NEJM199001113220205
28. Zahr NM, Kaufman KL, Harper CG. Clinical and pathological features of alcohol-related brain damage. Nat Rev Neurol. 2011 May;7(5):284-94. doi: 10.1038/nrneurol.2011.42. Epub 2011 Apr 12. PMID: 21487421; PMCID: PMC8121189.
29. Etminan N, Rinkel GJ. Unruptured intracranial aneurysms: development, rupture and preventive management. Nat Rev Neurol. 2016 Dec;12(12):699-713. doi: 10.1038/nrneurol.2016.150. Epub 2016 Nov 3. Erratum in: Nat Rev Neurol. 2017 Feb 1;13(2):126. doi: 10.1038/nrneurol.2017.14. PMID: 27808265.
30. Endrit Ziu; Mahammed Z. Khan Suheb; Fassil B. Mesfin. et al. Subarachnoid Hemorrhage. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024.
31. D'Souza S. Aneurysmal Subarachnoid Hemorrhage. J Neurosurg Anesthesiol. 2015 Jul;27(3):222-40. doi: 10.1097/ANA.0000000000000130. PMID: 25272066; PMCID: PMC4463029.
32. Grant BF, Chou SP, Saha TD, et al. Prevalence of 12-Month Alcohol Use, High-Risk Drinking, and DSM-IV Alcohol Use Disorder in the United States, 2001-2002 to 2012-2013: Results From the National Epidemiologic Survey on Alcohol and Related Conditions. *JAMA Psychiatry*. 2017;74(9):911-923. doi:10.1001/jamapsychiatry.2017.2161